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THE SCIENCE OF CHEMOTHERAPY

JOHN A. KOLMER, M.D.

Professor of Medicine, Temple University; Director of the Research Institute of Cutaneous Medicine

PHILADELPHIA

In 1867, when the work of Pasteur suggested to Lister that pus in a wound might be due to "fermentation" by some invisible living agent and that suppuration could be prevented by operating under a spray of phenol and by the application of antiseptic dressings, the era of antiseptic surgery began and the science of chemotherapy had its birth. In other words, even before 1879, when staphylococci and streptococci were discovered, Lister learned empirically how to destroy them and other microorganisms without serious damage to the host by local application of chemical agents, and today every time a surgeon applies tincture of iodine to the skin or treats local infections by topical application of chemical agents, he is practicing chemotherapy in that he seeks to destroy organisms by these means without damage to the tissues of the host.

The discovery of pathogenic organisms and the finding that various chemical agents were highly destructive for them in the test tube aroused hopes for a specific treatment of tuberculosis and other systemic infections. These expectations were soon dashed on the rocks of disappointment; for substances highly bactericidal in vitro were found ineffective in vivo because of a lack of specific parasiticidal effect and because of high toxicity for the host. Ehrlich, however, long entertained the idea that since lead and other chemical agents have a selective affinity or toxicity for certain body cells, it might be possible to produce some synthetically that would possess a selective affinity or toxicity for the living agents of disease and permit parenteral administration of amounts sufficient to exert specific parasitotropic or curative effects with little or no toxic or organotropic effects on the body cells of the host. This idea and dream were greatly strengthened by the discovery of antibodies, since here were chemical substances of biologic origin which specifically and directly attacked pathogenic bacteria or their products without damage to the host. In other words, Ehrlich thought that it was possible

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to find chemical agents in the mineral or vegetable kingdoms, or to produce some synthetically, which, while possessing sufficiently high toxicity or parasitropism for pathogenic organisms to be used in the specific treatment of infection, would yet be of sufficiently low toxicity or organotropism for the body cells of the host to permit their safe administration.

However, it was not until 1902, when Laveran and Mesnil succeeded in producing experimental trypanosomiasis in animals and tried a large number of chemical agents in its treatment, that modern chemotherapeutic investigation began in earnest. The new impetus resulted in the discovery of the trypanocidal activity of sodium arsanilate by Thomas and Breinl in 1905. In the same year Spirochaeta pallida was discovered by Schaudinn; a year later Parodi found that syphilis was transmissible to rabbits. These discoveries focused attention on trypanosomiasis, syphilis and other spirochetal infections and greatly renewed the hope of Ehrlich that it might be possible by chemical manipulations in the laboratory to produce new organic compounds of arsenic possessing greater parasitotropic or curative and lesser organotropic or toxic effects. This led him and his colleagues, Bertheim and Hata, to synthesize and try a large number of new compounds in the treatment of experimental trypanosomal and spirochetal infections. Their efforts culminated in 1909 in the discovery of arsphenamine, the greatest triumph in chemotherapy until the discovery by Domagk, in 1935, of the remarkable effects of the original prontosil (hydrochloride of 4-sulfamido-2′,4′-diaminoazobenzene) in the treatment of experimental streptococcic and other bacterial infections.

In other words, chemotherapy is not merely the treatment of disease with chemical agents; that is as old as the art of medicine itself. Rather it is the treatment of diseases produced by living pathogenic organisms with specific chemical agents or drugs capable of selectively destroying them without serious toxic effects. It is significant that in spite of constant effort to find specifics since the dawn of medicine, until the discovery of arsphenamine about thirty years ago, only three were known, namely, mercury for syphilis, quinine for malaria and ipecac for amebic dysentery. Chemotherapy is therefore the youngest of the therapeutic sciences, but already it has accomplished much with brilliant success in so many directions, with so much ground remaining to be explored, that its ultimate triumphs cannot be predicted. Suffice it to state that the scope of chemotherapy is extremely broad. It includes not only the destruction of pathogenic bacteria on the skin and mucous membranes and in wounds by topical application of chemical agents but also the prevention and treatment of bacterial diseases by oral or parenteral administration of such agents, as well as the prevention and treatment of syphilis and other spirochetal maladies, trypanosomiasis, malaria and

other protozoal diseases, and helminthiasis and other diseases due to animal parasites. Furthermore, a beginning has been made in the chemotherapeutic treatment of some of the viral diseases, especially if trachoma can be included in this category. Success has not yet attended the chemotherapy of the rickettsial diseases. Without reasonable doubt, however, success in the treatment of these diseases is to be expected in the future.

It was but natural for Ehrlich to swing his side chain theory of immunity into an explanation of the parasitotropism of chemical agents on the assumption that they selectively combine with organisms through the action of specific chemoreceptors. This idea is no longer seriously considered. It is now surmised that the chemical agent effects a union with one or more of the chemical constituents of the organism, resulting in the latter's crippling or death. However, comparatively little of a definite nature is as yet known of the laws of the constitution of chemical agents in relation to their parasiticidal or disinfective effects. There is much evidence to indicate that arsphenamine, bismuth and mercury are directly destructive of S. pallida in the test tube and in the tissues, and the same may be true of quinine for the plasmodia of malaria, but it cannot be denied that these compounds may injure the organisms only to the extent of reducing the proliferative activities, the final disposal of the invaders being effected through the immunologic activities of the body cells. Certainly, while the mechanism of the curative properties of sulfanilamide and its derivatives in bacterial diseases has not been definitely established, there is a large amount of evidence to indicate that these drugs produce bacteriostasis by crippling the proliferative activities of bacteria and that the disposal of the organisms is then achieved through immunologic processes, particularly through phagocytosis on the part of the fixed and wandering cells of the reticuloendothelial system.

Thus there is apparent an important relationship between chemotherapy and immunity so far at least as the bacterial infections are concerned. In other words, natural and acquired antibodies may play an important part in treatment and in the mechanism of curative activity, especially the opsonins, so essential in phagocytosis. For this reason I am still much in favor of chemoserotherapy or combining the administration of sulfanilamide and its derivatives with the appropriate use of immune serums (antimeningococcus, type-specific antipneumococcus, antianthrax), as well as of blood transfusions, in the treatment of severe infections, particularly those with which there are positive blood cultures or other evidences of septicemia. Furthermore, the administration of antitoxins is frequently required, especially in the treatment of scarlet fever and gas gangrene, for it is likely that none of the present chemotherapeutic agents, including sulfanilamide and its derivatives, are destructive of bacterial toxins, although they may reduce the production of toxins. It appears to me, then, that sulfanilamide and its derivatives

are not always a short cut or royal road to success in the treatment of severe infections and that physicians cannot afford to neglect or overlook biotherapy and adjunct measures which have served them so well in the past.

As previously stated, the laws of the chemical constitution of compounds in relation to disinfection are not yet well established. For this reason chemotherapeutic investigation is still largely on the "hit or miss" basis. Compounds of interest and promise should be studied for toxicity in animals, not only as to the maximum tolerated dose per kilogram of weight but also as to possible pathologic changes in the important organs, with special reference to the liver, the spleen and the bone marrow. Whenever possible, their curative activity should be determined in experimental infections of animals, as it is not at all likely that they will prove effective in human diseases unless they demonstrate some curative activity in experimental infections. It is true that animals do not always reveal toxic effects and that the toxicity for a healthy animal may be lower than that for a sick human being; then, too, it is not always possible to produce a satisfactory experimental infection. Nevertheless, tests for toxicity and curative activity in animals are of definite value and make it possible to obtain the chemotherapeutic index of the compound.

In other words, when new compounds are synthesized, they should not be administered to human beings until some adequate idea of their toxicity has been gained by oral or parenteral administration to animals. With such information at hand, one is justified in using a compound empirically before its therapeutic effects have been determined if the infection cannot be reproduced satisfactorily in animals; actual trial may be the only possible means of evaluation. Under the circumstances it is entirely likely that empiricism will continue to be an important and integral part of chemotherapeutic progress and under certain conditions is indispensable. Certainly no compound can be finally evaluated except by clinical experience.

In perhaps no other department of science is there psychologically so great a will to success as in chemotherapy. Throughout the literature compounds are hailed, by their discoverers at least, as the final solution of the problem in hand though in the light of more critical examination they may prove to be only of mediocre activity if not distinctly harmful. But merely to mention that empiricism alone gave us quinine for malaria, mercury for syphilis, ipecac and emetine for amebiasis, antimony and potassium tartrate for kala-azar and other forms of leishmaniasis and chaulmoogra oil for leprosy is sufficient to indicate the important role it has played in the development of chemotherapy.

Until the discovery of the uses of sulfanilamide and its derivatives in the treatment of bacterial and possibly some viral infections, chemo-

therapy had its greatest triumphs in the treatment of spirochetal diseases and those due to various protozoa and metazoa. Ehrlich always dreamed of synthesizing compounds that would be effective not only against a particular organism but against many. Too bad that this great investigator and benefactor of mankind did not live long enough to see his ideal largely realized in sulfanilamide and its derivatives! Until their discovery, the chemotherapy of the bacterial diseases was largely disappointing. Today their range of therapeutic effectiveness is so large and the research in the production of newer compounds so intense that the ultimate triumphs cannot be predicted although easily surmised. Unfortunately, however, the haphazard and indiscriminate administration of these new compounds for all and sundry infections is most undesirable; for there are still a number of infectious processes which do not respond to chemotherapy and in these prolonged administration may be not only useless but actually harmful.

It is difficult and sometimes impossible to determine on the basis of experimental and clinical data which of the various new compounds available is the one of choice in the treatment of a given bacterial infection, but opinion is rapidly crystallizing. For example, there can be no doubt that sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) is to be preferred in the treatment of staphylococcic infections, although for these chemotherapy is by no means as satisfactory as for hemolytic streptococcic, meningococcic and other infections. Furthermore, the sulfadiazine (2-sulfanilamidopyrimidine) synthesized by Roblin and his colleagues is apparently more effective than sulfapyridine (2-[para-aminobenzenesulfonamido]-pyridine) and sulfathiazole in the treatment of lobar pneumonia, and the introduction of sulfaguanidine (sulfanilylguanidine) by Marshall and associates is full of promise in the treatment of intestinal infections, especially in preparation for operations on the intestinal tract.

There can be no doubt about the effectiveness of local applications of sulfanilamide crystals in the prevention and treatment of infections of superficial wounds and burns and of local peritonitis. Indeed, it would appear that the local or oral administration of sulfanilamide and its derivatives for the prevention of infection is worthy of serious attention and wider application. This is true not only in relation to superficial wounds and burns but with regard to the prevention of puerperal sepsis, gas gangrene, epidemics of sore throat, the extension of hemolytic streptococcic infection to the middle ear and mastoid cells, peritonitis after injury or the rupture of appendical abscesses, infection after accidental pricking of the fingers in the operating or necropsy rooms, subacute bacterial endocarditis following the extraction of teeth in patients with valvular heart disease and the recurrence of rheumatic fever and streptococcic anginas as advocated by Thomas and her colleagues.

Fortunately, the toxic effects of sulfanilamide and its derivatives are generally recognized by physicians, and the dangers of self medication are well appreciated by the laity. However, we must distinguish complications due to disease and those due to treatment; many patients now live to present the former who without chemotherapy would have died before such complications develop. Of the true side effects of treatment with sulfanilamide and its derivatives, nausea with or without vomiting is particularly troublesome, and considerable attention has been directed to its causes and prevention. Evidence suggests that there is a local action, probably in the stomach, combined with a central action in the medulla. The quantity of sulfanilamide (or derivative) in the blood must be a factor in the production of general reactions, but present evidence seems to indicate that fluctuations in the blood levels are a more direct cause of distress than the absolute amount administered, since constant high levels are better borne than irregular lower ones.

A SIMPLE TECHNIC FOR PHOTOGRAPHY OF THE DRUM HEAD

IRVIN HANTMAN, M.D.
WASHINGTON, D. C.

In the past few years there has been a considerable increase in the utilization of color photography by members of the medical profession. In every specialty, particularly in those fields dealing with the body cavities, technics have been and are being evolved which best solve the particular problems that the photographing of these cavities presents. Because the photographic technician cannot be expected to understand the anatomic intricacies of some of these cavities, it has become necessary for the physician to help solve some of the technical problems involved.

The drum head in particular, because of its size and anatomic position, has proved itself a rather difficult structure to photograph. A review of the literature has failed to reveal any reference to photography of this membrane. However, recently the very obstacles encountered have apparently stimulated many otolaryngologists in that direction. In presenting an exhibit of photographs of the drum head at the Cleveland convention of the American Academy of Ophthalmology and Otolaryngology, I was surprised to learn of the number of investigators who have concerned themselves with this problem.

In the past year, in the Otolaryngologic Clinic of the Episcopal Eye, Ear and Throat Hospital, of Washington, D. C., I have been working along these lines, and at this time I wish to present a simple, convenient technic for photographing the drum membrane.

DESCRIPTION OF APPARATUS

An ingenious camera, made by the Cameron Surgical Specialty Company, has simplified the problem to a large extent, inasmuch as illumination, which represented one of the chief obstacles, is easily taken care of. A synchroflash bulb, delivering about 25,000 lumen seconds of total light output, is mounted within the camera, and when it is flashed, the light is thrown through a series of openings onto a silvered

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From the Otolaryngologic Clinic of the Episcopal Eye, Ear and Throat Hospital.

Presented at the Scientific Exhibit at the Forty-Fifth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, Cleveland, Oct. 6, 1940.

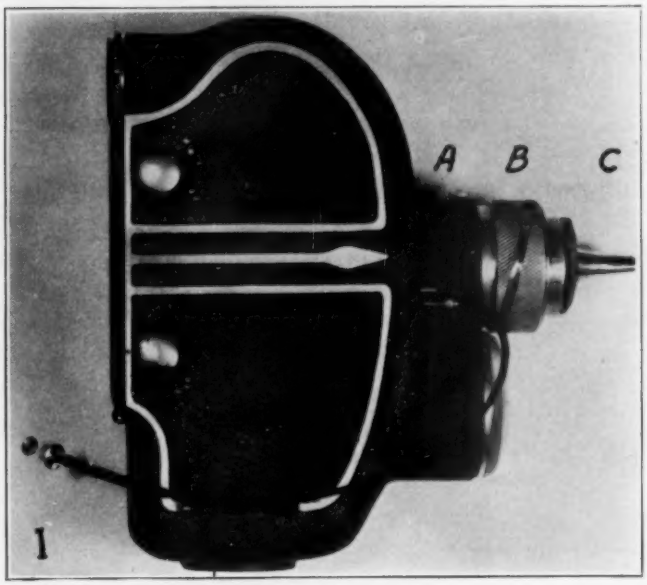
mirror, mounted at an angle of 45 degrees to the light source, and is projected forward into the cavity. In the center of this mirror is an achromatic lens of such fixed focal length that an object exactly 3 inches (7.62 cm.) from it is in focus, as seen through a ground glass mounted on the camera. At the moment the picture is snapped, the opening of the shutter and a flash explosion are synchronized. An image about the same size as a drum head is obtained.

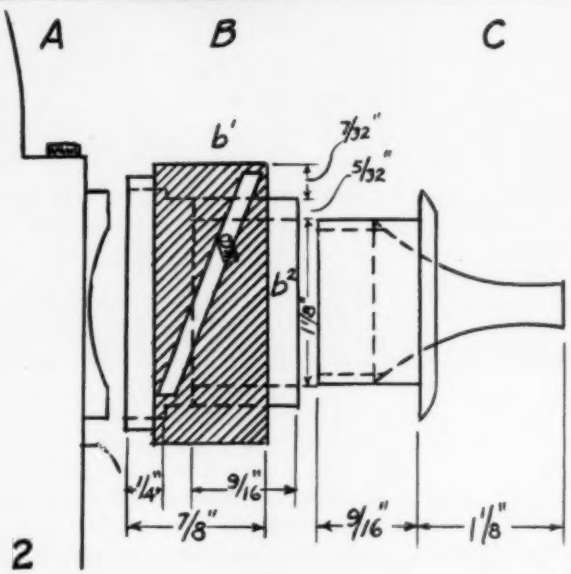
Because it is essential to obtain the membrane in exact focus, I have designed for use in conjunction with this camera a variable focusing device that attaches at its proximal portion to the camera and receives at its distal portion an ear speculum, which has been modified to fit the variable focuser (fig. 1). I have found it best to use the widest ear speculum that will fit into the canal, one with an oval cross section giving the best results. A large-sized Gruber or Toynbee speculum, prepared at its proximal end with dental compound to fit the variable focuser, has proved satisfactory. The focuser should be so constructed that it is not more than ⅞ inch (2.25 cm.) in length and capable of about ½ inch (1.3 cm.) increase in its projection. The part of the focuser that receives the speculum should be so made that although it moves the speculum forward and backward, it does not rotate the speculum in so doing. The speculum should be so mounted that the length of it plus the focuser is about 2 inches (5 cm.), capable of extension to about 2½ inches (6.3 cm.) (fig. 2). The inside of the speculum should be painted with a dull chimney black paint, to cut down reflections.[1]

TECHNIC

The ear speculum is inserted into the aural canal as deeply as possible without pain or discomfort to the patient, who is sitting in a chair as if for an examination of the ear, and the drum head is visualized with reflected light in as much of its entirety as possible. The camera, with the variable focuser mounted on it, is carefully slipped over the end of the ear speculum (an assistant steadies the camera by supporting it in the palm of her hand). A pilot light mounted within the camera provides the operator with enough light to visualize the drum head through the ground glass. The membrane is seen inverted and reversed, so that the cone of light is seen pointing posterosuperiorly instead of anteroinferiorly. By adjustment of the variable focuser the membrane is brought into exact focus, the handle of the malleus being used as the focusing point, and the signal is given to the assistant to release the shutter.

1. Modified forms of these attachments are made by the Cameron Surgical Specialty Company, Chicago.





Figs. 1 and 2.—*1*, photograph of apparatus for photographing the drum head. *2*, detail of variable focuser and modified ear speculum. In both figures *A* indicates the camera; *B*, the variable focuser, consisting of outside collar (b^1), (surface knurled), which when rotated moves the inside column (b^2), either forward or backward but does not rotate the speculum, and *C*, the modified ear speculum, the end of which is modified with dental compound to make a shoulder that will fit into b^2. This shoulder is slightly smaller than the inside of b^2, so that the variable focuser can be slipped over it easily.

Some care and practice will be necessary before good photographs are obtained. It takes a little experience to orient oneself while looking at the drum head reversed and inverted, but this is easily accomplished, as is the manipulation of the speculum and the camera. It is a good idea to take two or three exposures of the same ear drum at first, to insure getting a good picture.

Unfortunately, the technic presents certain disadvantages. The biggest difficulty is inherent in the size, shape and direction of the aural canal. In clinical examination of the drum head one is seldom able to see the entire structure at a glance without changing the direction of the speculum, so that actually the membrane is examined more or less piecemeal. However, a photograph of the membrane can take in only "one glance," as it were; therefore, in order that as much of the drum head as possible be included, it is necessary to have a wide external canal that is readily straightened to admit a large speculum.

I have found, also, that at times false color impressions, particularly of redness, are present on the colored film. This may be due to the fact that the light flash is too intense. It must be remembered, too, that in working at so close a range as 3 inches (7.62 cm.), depth of focus is slight, practically nil, and great care must be exercised to bring the drum head into exact focus before the picture is taken. I am now trying to obtain truer color and greater depth of focus by diaphragming the opening in the lens.

The most important advantage of this method is the simplicity and ease of application. Given a patient with a fairly wide aural canal, the picture can be taken within a few seconds, anywhere and at any time. As for expense, it is comparatively small considering this type of work. The cost of the camera is the largest item, but this camera can be used by the otolaryngologist for all types of photography, including that of the nose, throat and larynx, and also for exterior pictures. I do not believe that those interested in this procedure will complain of its cost.

SUMMARY AND CONCLUSIONS

A technic for photographing the drum head in natural color is presented, which is simple and convenient to perform. Not every drum head can be photographed, since it is necessary to have a wide external canal which is readily straightened out. However, with the proper selection of subjects and the refinement of technic which will readily follow practice, excellent photographs in color, showing fine details, may be obtained of the membrane, a structure which heretofore, because of its position and inaccessibility, has been difficult to photograph (fig. 3).

Farragut Medical Building.

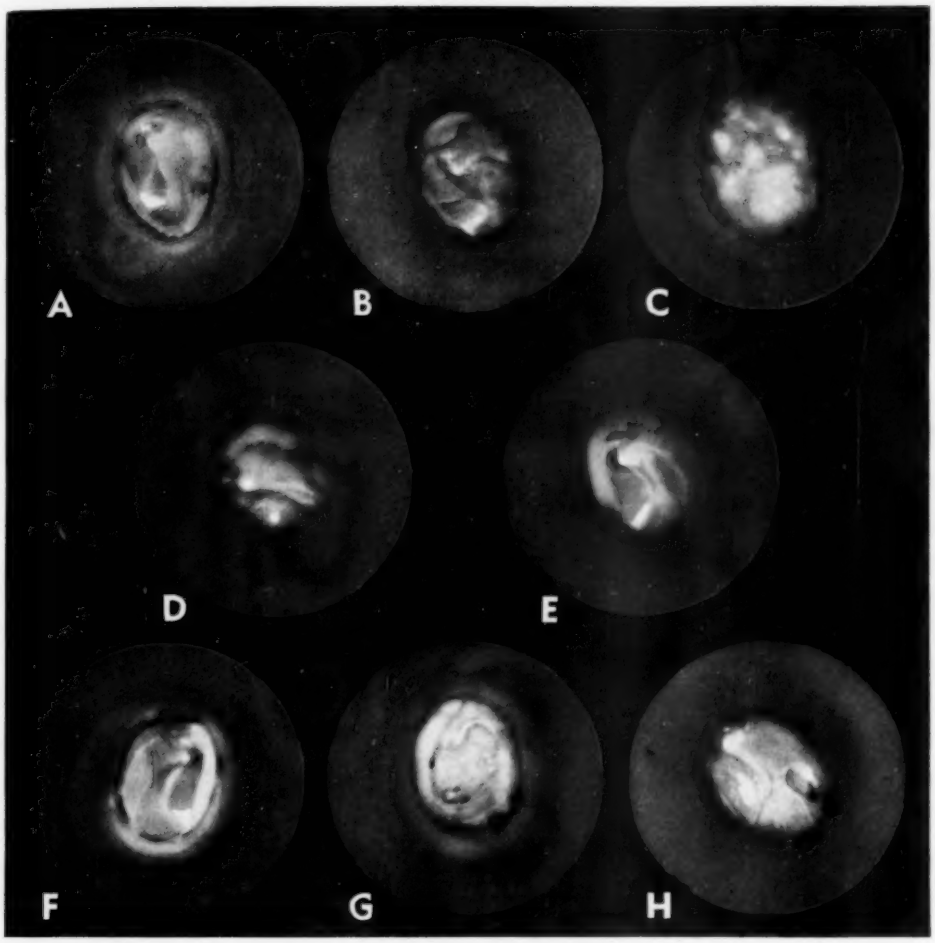


Fig. 3.—Photographs of the drum head in color (twice actual size): *A*, thin atrophic left drum membrane. The landmarks are easily visible. The long crus of the incus is seen shining through the posterior portion of the membrane. *B*, acute catarrhal otitis media in the left ear following an airplane ride. *C*, chronic otitis media in the left ear. The entire drum membrane is gone. One looks directly into the middle ear cavity. Note granulation tissue superiorly. *D*, left retracted drum membrane. The handle of the malleus is foreshortened. The membrane is very dull. *E*, normal drum head (left.). There is a slight false redness of the drum membrane superiorly. *F*, right drum membrane, showing early catarrhal congestion. *G*, large perforation of the right drum membrane. A remnant of the ossicle can be seen. *H*, left drum membrane, showing small posterior perforation and deposit of calcium anteriorly.

UNIV. OF MICH.

FRONTAL CELLS

AN ANATOMIC STUDY OF THESE CELLS WITH CONSIDERATION OF THEIR CLINICAL SIGNIFICANCE

O. E. VAN ALYEA, M.D.
CHICAGO

Considerable confusion exists in rhinology, and always has, concerning the classification of minor cells in the frontal area. These cavities have been variously termed "ethmofrontal cells," "anterior ethmoid cells" and "the frontal group of the ethmoid labyrinth." They have also on occasion been designated "duplicate" or "supernumerary" frontal sinuses. Certainly from a genetic and from a topographic standpoint the latter terms are well applied.

Davis [1] in his work on the development of the nasal sinuses reported supernumerary sinuses in 7 of 202 specimens. His observations also indicate that the frontal sinus is not always the earlier tenant of the frontal bone. A cell which causes a bulge in the floor of the sinus (frontal bulla) arrives at its mature position ahead of the sinus, which grows around it. Corroborative evidence of this may be found in certain adult specimens, those in which a cell is present in the posterior medial angle of the sinus; the frontal ostium, normally in this position, has been shifted to a point anterior to the cell (fig. 1; see also Van Alyea,[2] fig. 3 [suprainfundibular cell]). This would indicate that the cell was present in its adult position at some time prior to the invasion of the frontal bone by the sinus and that subsequent pneumatization carried the sinus beyond the cell. Cryer [3] cited instances of multiple frontal sinuses, as many as six in a single specimen. He justified his classification on the ground that cells which reach the squamous portion of the frontal bone, although small, have all the characteristics of the major

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Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc.

From the Department of Laryngology, Rhinology and Otology, and the Department of Anatomy, University of Illinois College of Medicine.

1. Davis, W. B.: Development and Anatomy of the Nasal Accessory Sinuses in Man, Philadelphia, W. B. Saunders Company, 1914.

2. Van Alyea, O. E.: Ethmoid Labyrinth: Anatomic Study, with Consideration of Clinical Significance of Its Structural Characteristics, Arch. Otolaryng. **29**:881 (June) 1939.

3. Cryer, M. H.: Some Variations of the Frontal Sinus, J. A. M. A. **48**:284 (Jan. 26) 1907.

cavity, which is called the sinus. Concerning a specimen which contained four cavities in the frontal bone, he said, "Some writers would class the two middle sinuses as anterior ethmoid cells which had invaded the frontal bone. If these cells should exist without the two larger sinuses they would then be called frontal sinuses by the same person." Kasper [4] in his study of 100 specimens found that reduplication of the frontal sinus was of common occurrence. He said, "These [cavities] are easily understood if one thinks of the frontal sinuses as ethmoidal cells growing beyond the confines of the ethmoidal mass into the various parts of the frontal bone." Schaeffer [5] also reported the presence of

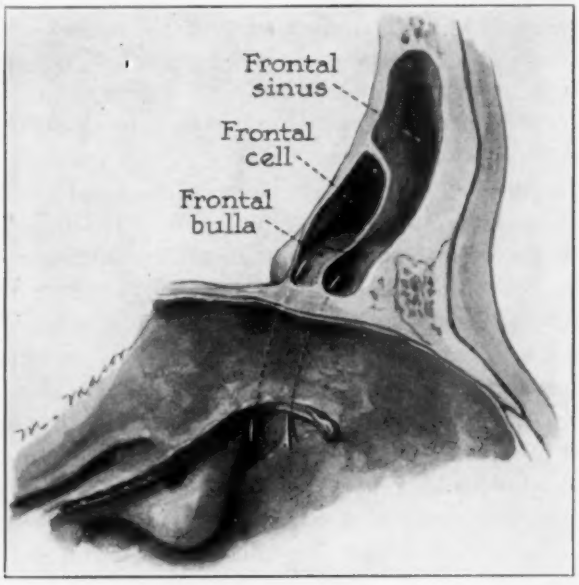

Fig. 1.—Nest of cavities. A large cell well within the frontal sinus is itself encroached on by a cell (frontal bulla) which causes an upward protrusion of the floor. The frontal ostium, normally in the medial posterior-inferior angle of the sinus, is shifted to an anterior position.

duplicate sinuses, although at times he designated these cells by other terms, including "frontal cell." This title seems to me a fitting appellation for certain types of minor cells in the neighborhood of the frontal sinus.

The cells referred to fall into two definite anatomic groups. They are: (1) frontal recess cells, those limited in origin and extent to the

4. Kasper, K. A.: Naso-Frontal Connections: A Study Based on One Hundred Consecutive Dissections, Arch. Otolaryng. **23**:322 (March) 1936.

5. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Naso-Lacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

frontal recess; (2) invading frontal cells, those which, regardless of origin, expand sufficiently to encroach on the area of the frontal sinus.

Rhinologic literature contains few articles which deal solely with frontal cells. When these are mentioned at all, it is usually with reference to a minor role which one of them may play in a frontal sinus infection.[6] Shea [7] mentioned this relationship and said, "The encroachment of a supraorbital cell elevates the floor of the frontal sinus to form the frontal bulla, narrowing the frontal duct." He also called attention to the possibility of duplication of the frontal sinus, which must be constantly remembered when one is establishing drainage, as each sinus communicates separately with the nasal cavity. Anderson,[8] in his observations on intranasal surgical treatment of the frontal sinus said, "A frontal ethmoidal cell frequently extends up over the orbit, and its superior wall forms the floor of the frontal sinus. This wall must be broken through if the frontal sinus is to be adequately drained." In a recent article Lillie and Simonton [9] called attention to the clinical potentialities of such a cavity when it encroaches on the lumen of the sinus. Without doubt many conditions due to such encroachment occur in rhinologic practice but remain unrecognized.

An anatomic investigation of the two types of cavities designated as frontal cells was deemed advisable because of the tendency of rhinologists to underestimate the clinical importance of such cavities or, as is often the case, to overlook them entirely.

Such an investigation was recently carried out on a series of specimens from cadavers at the University of Illinois College of Medicine. Skulls were taken at random from those available in the anatomy laboratory and in the collection of the department of otolaryngology. Specimens were examined one by one until 100 were found containing frontal cells of one type or another. To procure this number it was necessary to examine 242 specimens. Those without cells were excluded. In the study of the 100 remaining specimens an effort was made to classify frontal cells into anatomic groups based on the point of origin, location and structural characteristics and consider the clinical significance of the anatomic observations.

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6. Van Alyea, O. E.: Irrigation in the Treatment of Disease of the Frontal Sinus, Arch. Otolaryng. **19**:224 (Feb.) 1934.

7. Shea, J. J.: Morphologic Characteristics of the Sinuses, Arch. Otolaryng. **23**:484 (April) 1936.

8. Anderson, C. M.: Some Observations on the Intranasal Operation for Frontal Sinusitis, Minnesota Med. **18**:744 (Nov.) 1935.

9. Lillie, H. I., and Simonton, K. M.: Developmental Extension of an Anterior Ethmoid Cell Within the Frontal Sinus, Arch. Otolaryng. **32**:32 (July) 1940.

ANATOMIC GROUPING OF THE CELLS STUDIED

The cells in 56 of the 100 specimens fell into the first group, as frontal recess cells, and those in 63 were in the second group, as cells which invaded the frontal sinus. In 19 specimens both types of cells were represented.

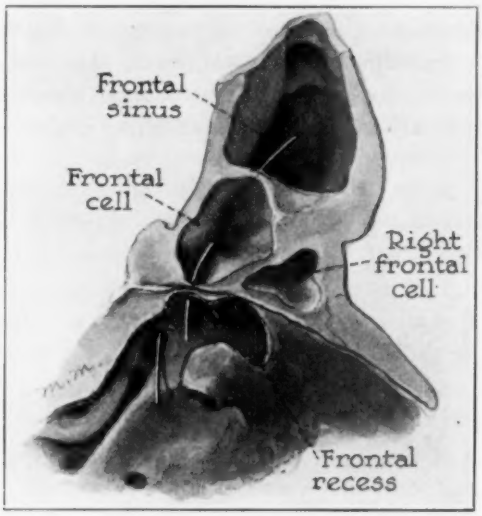

Fig. 2.—Intersinus cells and frontal recess extension. In the specimen illustrated the large cell in the medial wall of the frontal sinus materially constricts the lumen of the nasofrontal channel.

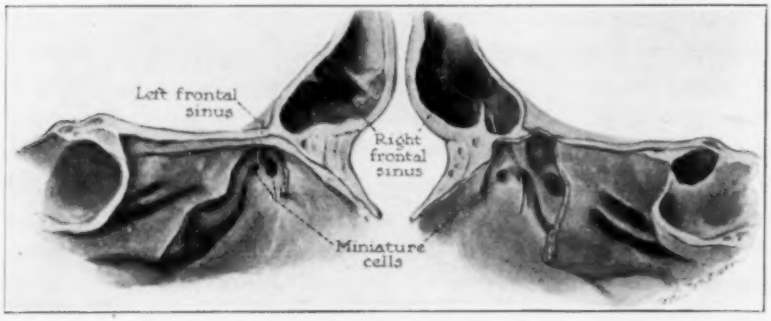

Fig. 3.—Miniature cells in the frontal recess. In the specimens illustrated (a bilateral study), there were two miniature cells on the left side and one on the right. In this skull the right frontal sinus excavated both frontal bones, the left frontal sinus being a small cavity posterior to the invading right frontal sinus.

FRONTAL RECESS CELLS

In the frontal recess, aside from the extension of cavities to the frontal bone, three types of cell expansion were noted. In 26 specimens

there was an anterior extension of the recess (fig. 2; see also Van Alyea,[2] fig. 10*a*). In these there was no discernible cell formation, and the excavated area opened widely into the middle nasal meatus. In 40 specimens small cells were present in the recess. These cells were reminiscent of the early buds which appear in this area in midfetal life. In the adult two distinct types may be recognized: the one, minute but well proportioned (figs. 3 and 4); the other, poorly developed and rudimentary (fig. 5). Those of the first type resembled miniature ethmoid cells and were present in 7 of the 40 specimens. These cavities were small in all dimensions, seldom measuring more than 3 or 4 mm. in depth or width, with an ostium only 1 or 2 mm. in diameter. In the remaining 33 specimens the cells were wide and shallow, being nothing more than slight depressions in the lateral wall of the recess. In some specimens the entire recess was involved in a single depression, while in others ridges were present which indicated dividing lines between cells. In 7 specimens combinations of cells were present, there being two or more varieties of frontal recess cells in each. Excavation of the frontal recess on rare occasions extended forward and downward beyond the attachment of the middle turbinate (fig. 2), and in a few cases the agger nasi was undermined by the pneumatizing process (fig. 6). A cavity of this type should not be confused with the true agger nasi cell, which always drains into the ethmoid infundibulum (see Van Alyea,[2] fig. 3).

INVADING CELLS

Cells arising in the frontal recess commonly extend upward to pneumatize the frontal bone and become the frontal sinus, or one of them may arrive at a point between the sinuses to become an intersinus cell (see Van Alyea,[2] figs. 2 and 11). In the latter event the cell usually encroaches on the medial wall of one or both of the frontal sinuses.

Other encroaching cells arise from the ethmoid infundibulum, ethmoid bulla and points above these structures. The largest number of invading cells are suprainfundibular in origin. Twenty-six cells of this type were noted in the present study. Invading cells which drained directly into the infundibulum numbered seventeen, while cells arising from the frontal recess were present in 15 specimens. Of cavities from the bullar areas, only 5 were found to encroach in any manner on the frontal sinus, and most of these also invaded the supraorbital plate of the frontal bone.

Invading cells vary considerably in size and extent. One cell may form only a slight bulge in a wall of the sinus, while another may project into the lumen almost to the extent of complete replacement of the sinus.

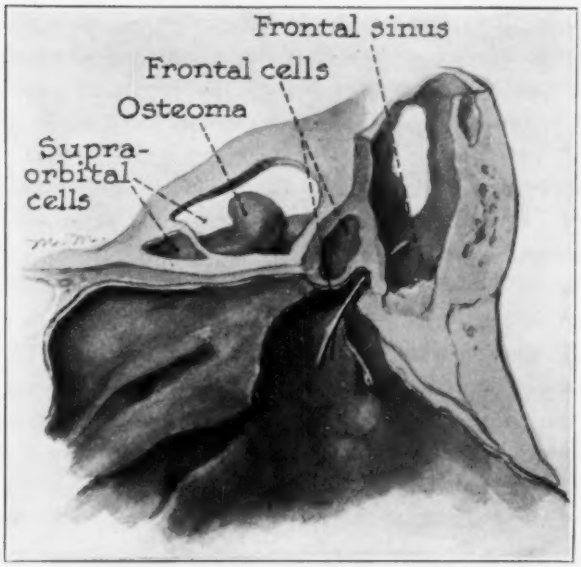

Fig. 4.—Supraorbital cells, a cell in the posterior wall of the frontal sinus and a miniature cell in the frontal recess. In this specimen two ostiums were found which had grown from the floor of a large supraorbital cell. A cell in the posterior frontal wall impinges slightly on the frontal ostium.

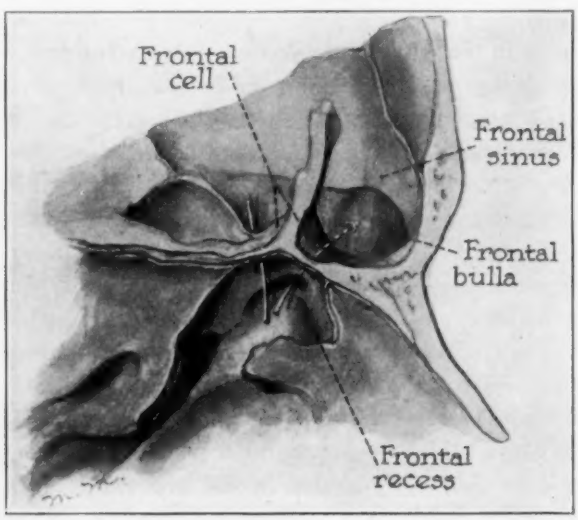

Fig. 5.—Cells in the frontal sinus and frontal recess. A large cell in the floor of the sinus extends laterally and posteriorly over the orbit. An excavation of the frontal recess extends above and beyond the agger nasi, and the depression in the wall is reminiscent of the rudimentary frontal cells of late fetal life. The frontal ostium is constricted by the invading cells.

Encroachment is not always made by a single cell. Among the 63 specimens containing invading cells there were 12 in which two such cavities were present. In one of these a nest of cavities was formed by a cell within a cell (fig. 1). The lower cavity caused an upward protrusion of the floor of the upper cell, which itself was located well within the lumen of the sinus. In another specimen a tier or nest of cells was present in the medial aspect of the sinus. Two large cells were present, one above the other; the lower cell undermined the agger nasi (fig. 6).

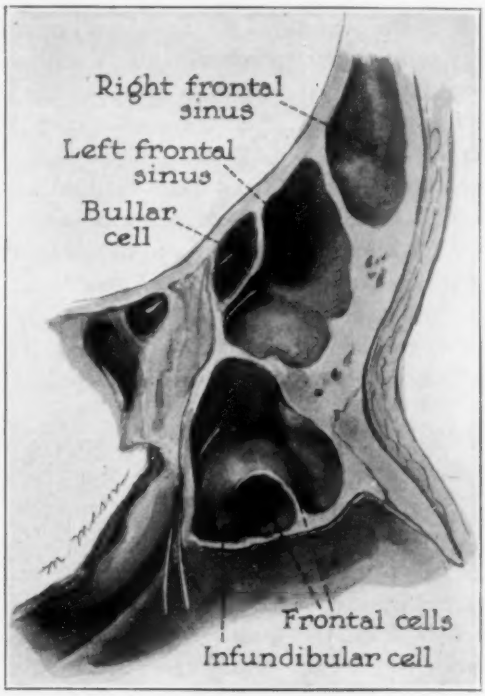

Fig. 6.—Tier of cells. The lower cell, which excavates the agger nasi, produces a bulge in the floor of the upper cell, which in turn causes a convexity in the floor of the frontal sinus. The upper cell, aided by an overhanging bullar cell, constricts the frontal ostium and contributes to the formation of a narrow tortuous nasofrontal duct.

CELL LOCATION

The expansion into the frontal sinus may be by way of the floor (frontal bulla), the posterior wall or the medial wall. Twenty-five cells were observed to encroach in some manner on the floor of the frontal sinus (figs. 1 and 6; see also Van Alyea,[2] fig. 3). When such expansion occurs, the floor of the sinus, normally concave, assumes a convexity,

which may involve its entire extent or which may be limited in size to an elevation 5 to 10 mm. in diameter. When a cell protrudes into the lumen of the sinus it has more the appearance of an aberrant ethmoid cell, being a round thin-walled cavity of medium size (fig. 5). The larger cell which displaces the medial and posterior walls has a thick wall and often extends laterally over the orbit (fig. 1).

The medially placed (intersinus) cell is the most frequent in occurrence of all the encroaching frontal cells. It varies considerably in size and extent of encroachment on the frontal sinus. In some instances a cell has been uncovered between the frontal sinuses which caused no perceptible change in the contour of the septal wall of either cavity. In most cases, however, a bulge is produced in this wall on the side from which the cell originates. The expansion may be sufficient to reach or pass beyond the frontal ostium; then the encroaching cell may assume a role of considerable clinical importance.

The cell in this area, whether from the medial or from the posterior wall, may interfere with the drainage of the frontal sinus by overhanging the ostium and by constricting and adding to the tortuosity of the nasofrontal channel (figs. 1, 2, 5 and 6; see also Van Alyea,[2] fig. 2).

The intersinus cell occurred in 28 specimens in the present study. In 14 of these it drained into the frontal recess; in 11, into the area above the infundibulum, and in the remaining 3, into the infundibular groove.

A cell encroaching on the posterior wall was present in 21 specimens. Often it was a small cavity at some distance from the frontal ostium. Such cells add to the variety of the relief in the sinus wall but aside from that are apparently of little clinical importance. Of more importance is the large cell in the posterior wall near the midsaggital plane. Often it occupies the position which is the usual domain of the frontal ostium, and when it does, the drainage point of the sinus is located immediately anterior to the encroaching cell (fig. 1).

SUPRAORBITAL CELLS

A cell which invades the supraorbital plate of the frontal bone commonly presses forward to communicate with or displace the posterior wall of the sinus (fig. 4; see also Van Alyea,[2] fig. 7). A common wall exists between these two cavities, and occasionally a communicating ostium is present in this wall. In a case in the present series the frontal sinus could be reached only by way of this opening. The sinus drained into the supraorbital excavation, which in turn drained into the middle meatus through the bullar ostium.

The supraorbital cell is, in most instances, an extension from the ethmoid labyrinth. It may arise from the infundibular, bullar or pos-

terior area. Rarely does a cell originating in the frontal recess reach the supraorbital plate and then only by first traversing the frontal sinus. One such cell was observed, a large cavity which entered the sinus at the medial border extended laterally along the floor and then passed through the posterior wall to become flattened out over the orbit. Fourteen supraorbital cells were noted in the present series, one less than those recorded in a previous study of a like number of specimens.[2]

COMMUNICATING CELLS

Not infrequently a frontal cell is found that communicates with the frontal sinus. In 10 of the 63 specimens containing invading cells, such a relationship was observed. The communicating link was at times a large space which took up almost the entire width of the cell.

Although the two cavities may be connected by an opening, each may have its own separate drainage outlet into the middle meatus. In that event, the two cavities are afforded better drainage facilities. For the same reason the two ostiums must also be regarded as two separate avenues through which infection may travel to the communicating cavities. In many cases, however, the two cavities utilized a single outlet. This was always a wide space, apparently the combined width of two ostiums.

COMMENT

Cells limited to the frontal recess in origin and extent, although comparatively common (they are found in approximately 50 per cent of all persons), probably seldom, if ever, attain any clinical importance. With the exception of the minute cells, which are rare in occurrence (approximately 6 per cent), drainage is through wide areas directly into the middle meatus.

The mature frontal cells are important in clinical rhinology. Because of their apparent insignificance, they often fail to receive the consideration they merit. Rhinologists customarily regard these cells as ethmoidal and include them in the anterior group along with the infundibular and bullar cells. The fallacy of this classification becomes apparent in cases of an isolated infection of one of these cells and in cases of cell infection associated with disease of the frontal sinus.

The consideration of the relationship of an invading cell to the frontal sinus is of much more importance clinically than that of one of these cells as a locus of infection. Encroaching cells are present in some form or other in approximately 50 per cent of all persons and in about a third of these the encroachment occurs in the neighborhood of the frontal ostium. That this influences drainage of the sinus in many instances is an unquestionable fact.

DIAGNOSIS

The symptoms of disease of a frontal cell are similar to those of frontal sinusitis. Delineation of the cell is possible by roentgen examination (fig. 7). Probing the frontal area may prove a valuable diagnostic

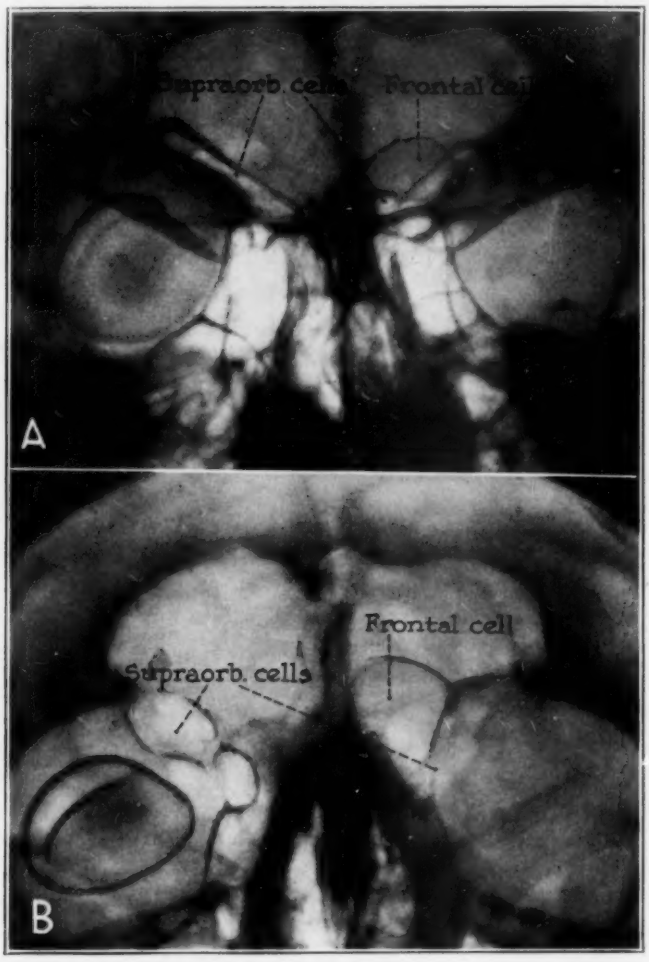

Fig. 7.—Roentgenograms showing an invading cell in the floor of the left frontal sinus (frontal bulla) and supraorbital cells on both sides. (The right eye is artificial.)

aid. A probe or cannula is passed into the frontal area as far as possible. If the lumen of the sinus has been reached, the cannula may be rotated, and its shaft rests against the patient's chin. When the tip of the instrument is in a cell, its movement is limited, and the shaft protrudes

at an angle from the chin. Roentgenograms with the cannula in place, taken in both lateral and frontal views, reveal the location of the tip. When this is known, diagnostic lavage is of great value. Further aid may be obtained with the use of radiopaque oil.

TREATMENT

Rhinologists who practice conservatism in sinus therapy hesitate to resort to the external operation on the frontal sinus when other methods will suffice. Most attacks of frontal sinusitis clear up spontaneously within a week or two with no treatment at all, and many require little more than palliative measures to effect a cure. There are certain ones, however, which are not so easily treated. These, on the contrary, are often a challenge to rhinologic skill. Included in this group are the acute sinusitis with severe symptoms, the persistent acute, the subacute, the recurrent and the chronic involvement and the vacuum and latent type of infection.

The major concern of the rhinologist in dealing with these refractory conditions is the problem of sinus drainage. Completely or partially blocked nasofrontal passageways account in large measure for the presence and persistence of frontal sinus infections, and this should be kept in mind when outlining a therapeutic regimen. Treatment should be directed first toward the relief of symptoms and the elimination of the attack. Later, between attacks, the question of prevention of recurrence may be considered.

Irrigation is advocated, along with other measures, in the treatment of most stages and types of disease of the frontal cavities. This method of therapy has proved to be a comparatively safe and effective one for affording early relief and combating the disease. The frontal sinus is more responsive to irrigation than the maxillary. This is due in part to the fact that in the majority of cases the frontal ostium is located in the most dependent point of the sinus floor and drains through a wide orifice directly into the frontal recess. Also, in the case of the frontal sinus, the results obtained by lavage are due not merely to the removal of purulent material but to the restoration of the natural pathways of drainage as well. Often, in the passage of a frontal cannula, a thin-walled frontal cell is encountered in the neighborhood of the ostium. The wall is easily broken down by the tip of the cannula, and the nasofrontal passageway is thereby enlarged. The trauma caused to the lining of the sinus and the ostium by the passage of the cannula is more than offset by the improvement in the drainage facilities.

Cells with thicker walls may be obliterated by use of a small nasal rasp. This instrument is used only in selected cases and then with

extreme caution. In the treatment of subacute and early chronic sinusitis a slight enlargement of the frontal ostium will often aid materially in effecting a cure. For the recurrent type this procedure may be performed between attacks. Rasping a frontal ostium is not a common practice of rhinologists. Many hesitate to adopt it because they feel that the resultant granulation tissue offsets whatever benefit may be gained by the operation. They also fear the development of osteomyelitis. The latter complication is less likely to occur following the use of the rasp on the compact bone of the sinus floor or the wall of a frontal cell than following operation on the anterior sinus wall.

In many cases disturbance in frontal drainage is not due to a narrow ostium but to a sealed middle meatus. Removal of the block in the middle meatus may be accomplished by infraction of the middle turbinate or by crushing of a cellular turbinate. These measures may be most effectively preceded by resection of a deviated nasal septum. Removal of any portion of the middle turbinate is discouraged. Destruction of normal functioning turbinate tissue is an unnecessary, harmful procedure, and it is of questionable value in improving sinus drainage.

The external frontal operation is rarely found necessary or advisable. It is reserved for the severe sinusitis with complications or impending complications, the hyperplastic condition and the persistent chronic involvements which resist all other forms of treatment.

SUMMARY

Considerable confusion exists concerning the classification of minor cells in the frontal area. The term "frontal cells" is regarded as a fitting title for these cavities because topographically, clinically and, in many instances, genetically they are more frontal than ethmoidal; yet they should not be confused with the major cavity of the frontal bone, the frontal sinus.

Frontal cells may be grouped into two definite anatomic types: frontal recess cells and invading frontal cells. The invading type is important clinically, not only as a possible locus of infection but also because of its influence in drainage of the frontal sinus.

Because of their apparent insignificance the importance of these cells is often underestimated, or their presence is overlooked completely. They occur, however, with sufficient frequency to justify consideration of their presence as a possible entity in any study of frontal sinus disease.

In a recent examination of a large series of specimens containing frontal cells, from adult cadavers, the frequency and nature of cell encroachment on the frontal sinus were revealed. Frontal cells impinging on the frontal sinus appear in some form or other in approximately 50

per cent of all persons, and in approximately one third of these the invasion is in the neighborhood of the frontal ostium. That the presence of such a cell influences drainage of the sinus in many instances is an unquestionable fact.

The symptoms of disease of a frontal cell resemble those of frontal sinusitis. Roentgen study with or without the use of radiopaque oil is of considerable aid in diagnosis (fig. 7). Probing or cannulation of the frontal area is important, not only as a diagnostic but also as a therapeutic measure. Irrigation is advocated as a comparatively safe and effective therapeutic procedure in most types of disease of the frontal cavities. Passage of a cannula for this purpose often breaks down a thin-walled cell adjacent to the frontal ostium, and the nasofrontal passageway is thereby enlarged.

A cell with a thicker wall in this area may often be obliterated by use of a small nasal rasp. Also, enlargement of the frontal ostium with this instrument is advocated in selected cases. Infraction of the middle turbinate, crushing of a cellular turbinate and resection of the nasal septum are effective in certain cases.

The external frontal operation is rarely found necessary, or advisable. It is reserved for the severe sinusitis with complications present or impending, the hyperplastic condition and the persistent chronic involvements which resist all other forms of treatment.

OTITIS AND AIR CELL SYSTEMS

MARCUS DIAMANT, M.D.
HALMSTAD, SWEDEN

In the last few decades it has frequently been asserted that a connection exists between the size of the air cell system in the temporal bone, on the one hand, and the development and course of otitis, on the other. The earlier view that chronic otitis results in a sclerosing of the bone attended by the disappearance of a previously existing cell system has been abandoned. Heine [1] and Cheatle [2] were the first to communicate observations on the presence of a sclerotic mastoid bone without coexistent otitis. That a sclerotic mastoid bone is so often found in cases of chronic otitis is due, in their opinion, to the fact that deficient pneumatization favors the development of chronic otitis.

Investigations so far carried out show that in different persons the air cell system varies in size within fairly narrow limits. The size of the cell system has been estimated by macroscopic examination of preparations or of roentgenograms, and on the basis of such estimates the material of investigation has been divided into groups. Thus, the size of the individual cell systems has not been determined by exact measuring. The variability in size and the frequency of the different sizes in the normal material therefore could not be established. This applies also to the pathologic material. The absence of exact data on the normal material naturally renders an estimate of the pathologic material more difficult.

Investigations on material from patients with chronic otitis have shown, however, that this disease developed chiefly in ears with comparatively small air cell systems. It has not been possible by approxi-

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This communication is a review of the monograph by Marcus Diamant "Otitis and Pneumatisation of the Mastoid Bone," issued from the State Institute of Human Genetics and Race Biology, Uppsala, Sweden, in 1940, Prof. Gunnar Dahlberg, M.D., LL.D., director (Acta oto-laryng., 1940, supp. 41, pp. 1-149).

1. Heine, B.: Operationen am Ohr, Berlin, S. Karger, 1904.

2. Cheatle, A. H.: The Infantile Types of the Temporal Bone and Their Surgical Importance, Lancet **1**:491, 1910; Twenty Specimens of Chronic Middle Ear Suppuration and Its Sequelae, Proc. Roy. Soc. Med. (Sect. Otol.) **3**:41, 1910; Three Specimens of Chronic Middle-Ear Suppuration in Each of Which the Opposite Side Was Normal, the Six Bones Being All of the Diploëtic Infantile Type, ibid. **4**:112, 1911; An Examination of Both Temporal Bones from One Hundred and Twenty Individuals Made with the View of Deciding the Question of Symmetry, ibid. **6**:59, 1913; The Etiology and Prevention of Chronic Middle-Ear Suppuration, Acta oto-laryng. **5**:283, 1923.

mate estimates of the sizes of air cell systems to establish an upper limit at or above which chronic otitis does not develop. Neither has it been possible, within the group of "small" cell systems, to distinguish between different sizes and to establish a potential connection either with different forms of chronic otitis or with different changes of permanent nature in the tympanic membrane.

Workers on material from subjects with acute otitis maintain that the disease develops in ears with large air cell systems. Some writers are of the opinion that this applies not only to cell systems which are large as compared with those found in persons with chronic otitis but to large cell systems generally. No definite conclusions on this point can be reached by estimates of the sizes of air cell systems.

In 1918 Wittmaack [3] published his large work on normal and pathologic pneumatization and the connection of the latter with otitis. His investigation did not directly refer to the size of the air cell system and its connection with the development and course of otitis. He tried to show the significance of the mucous membrane (the mesoplastic, hyperplastic and hypoplastic types) for the development both of air cell systems and of otitis. He stated that a roentgen examination of the air cell system made it possible for him to draw conclusions with regard to the type of mucous membrane present which otherwise could not be established without operation and microscopic examination. He himself showed, however, that the different pathologic types of mucous membrane are present with different sizes of cell systems, ranging from the smallest toward the largest. On the other hand, according to Wittmaack, with "small" cell systems pathologic types of mucous membrane are always present, but on this point, too, contradictory statements are found in the literature. Thus, the size of the air cell system can hardly give a definite clue to the type of mucous membrane present. Though Wittmaack referred to the size of the air cell system, the lack of exact measurements naturally renders more difficult a definite solution of the problems in his investigations.

THE PROBLEM

The problem of the present investigation was to compare by more exact methods the sizes of the air cell systems in mastoid bones as they appear in roentgenograms of normal persons and in those of persons with otitis of various types. Comparisons were made both between persons in the different age groups and between the two ears of the same person, thus referring also to conditions of age and of asymmetry.

3. Wittmaack, K.: Ueber die normale und die pathologische Pneumatisation des Schläfenbeines; einschliesslich ihrer Beziehungen zu den Mittelohrerkrankungen, Jena, Gustav Fischer, 1918.

MATERIAL AND METHOD

The material comprises roentgenograms of 905 persons at different ages, 320 of whom were normal. The other 585 had otitis of different types; about 50 per cent of them had chronic otitis as defined by Brock [4] in the textbook of Denker and Kahler.

Both ears of all the persons included in the study (i. e., 1,810 ears) were examined clinically and roentgenologically by current methods of investigation. Otoscopic examination was made with the von Eicken-Leitz [5] binocular magnifier, and roentgenography was done with the use of the Lysholm [6] skull table. The determination of the size of each air cell system was based on a roentgenogram showing the extension of the cell system in lateral projection, the central ray from the focus to the ear forming an angle of 35 degrees with a horizontal plane through both ears (axial orthoprojection in lateral position, or P_{II}, according to Lysholm and to Runström [7]). It was shown by special examinations and by calculations of correlation coefficients that the size of the air cell system as it appears in lateral projection is fairly representative of the volume of the cell system.

The size of the cell system in the roentgenogram was obtained in square centimeters by measuring with a planimeter. This instrument renders possible a quick and easy measuring even of irregularly demarcated surfaces.

The sources of error inherent in both the roentgenologic and the planimetric method were determined and proved to be insignificant. Thus if roentgenography is done with use of the Lysholm skull table in the given projections, the pictures when measured with a planimeter will give fairly corresponding values even for different investigators. The numerical measuring values presented in different tables and obtained by special determinations of standard errors give information regarding the limits within which the results obtained by other workers under the given conditions may deviate from the ones obtained in the present investigation. Scientific investigations bearing on the size of the air cell system thus may be checked by the employment of these methods, and the results of those carried out in the same manner may be directly compared.

ANALYSIS OF OBSERVATIONS

Examination of the material from normal subjects at different ages shows that the development of the air cell system takes place mainly in early childhood. Between the ages of 10 and 15 years a sex difference in the rhythm of growth has been established, as growth seems to be terminated earlier in females than in males. It is easily imagined that this dissimilarity corresponds to other differences in the rhythm of growth in the two sexes just before and during the years of puberty.

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4. Brock, W., in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohren- heilkunde, Berlin, Julius Springer, 1926, vol. 6.

5. von Eicken, C.: Meine Binokularlupe für Hals-, Nasen- und Ohrenärzte, Acta oto-laryng. **5**:418, 1923.

6. Lysholm, E.: Apparatus and Technique for Roentgen Examination of the Skull, Acta radiol., 1931, supp. 12, p. 1.

7. Runström, G.: A Roentgenological Study of Acute and Chronic Otitis Media, Acta radiol., 1933, supp. 17, p. 1.

The secondary sex characteristics, for instance, appear earlier in females.

The present investigation also shows that one has to reckon with a moderate regression of the size of the air cell system after the age of 30. This regression can be explained by the assumption that the osteoblastic and osteoclastic bone processes continue through the entire lifetime and, with regard to intensity, an imbalance develops in the direction of increased new formation of bone, by which the size of the cell system decreases.

The investigation shows that the measured size of the cell system as shown in the roentgenogram made in the given projection normally varies in adults from 0 up to about 28 to 30 sq. cm., with an average size of about 12 to 13 sq. cm. The distribution of the different sizes in normal subjects shows good correspondence with a normal curve.

The average values for the air cell system given here refer to the age group 10 to 30 years.

The examination of patients with chronic otitis shows that the average values for the size of the air cell system (2.89 ± 0.27 sq. cm.) differ throughout, in a statistically significant manner, from the values of normal subjects (12.27 ± 0.40). Ears presenting chronic otitis have smaller air cell systems throughout. On the whole I may state that the difference amounts to about 75 per cent of the average value for the normal material. Thus, patients with chronic otitis on an average have one-fourth as large air cell system as normal persons have. In particular it should be pointed out that in practically every case of chronic otitis the size of the cell system falls short of the average value for normal subjects. In other words, if, by the methods here described, a cell system is found to exceed 15 sq. cm. in projection P_{II}, one may assert that otitis, if it should occur, will not give rise to a permanent change of the tympanic membrane. This means that the person will never have chronic otitis. Apart from the prognostic value of this piece of information, a greater significance should perhaps be ascribed to the fact that one's conception of the causes of chronic otitis can now be clarified by the knowledge that factors determining the development of the disease are definitely correlated with the size of the air cell system. The question whether neglect of treatment of acute otitis leads to chronic otitis thus has to be examined again. The present investigation does not show that such neglect is not the cause of the development of permanent changes in the tympanic membrane in the presence of a small air cell system, but it indicates that not even neglect of treatment can result in chronic otitis if the cell system exceeds the average value for normal subjects. The same is true of scarlatina as a cause of chronic otitis.

The patients with chronic otitis have been divided into two main groups including those with marginal and those with central perforations respectively. The cell systems of patients with chronic otitis with marginal perforation present an average value amounting to 1.81 ± 0.33 sq. cm. Patients with chronic otitis with central perforation of the tympanic membrane have cell systems the average measurement of which is 4.15 ± 0.52. The difference between these two is statistically significant. It means that different factors are actually represented in the sizes of the cell systems, determining the development on the one hand of marginal perforation, on the other of central perforation. (It should be remembered that the cause of central perforation of the tympanic membrane is well known, but the cause of marginal perforation is more or less unknown.)

It is a well known fact that chronic otitis may heal, with an atrophic scar formation covering the previous perforation. Measurement of the cell systems of ears with atrophic scar healing of the tympanic membrane yield an average value of 5.26 ± 0.62 sq. cm. This value shows a statistically significant difference as compared with the average value for ears with open perforations. Thus in the presence of a sufficiently large cell system (exceeding 15 sq. cm.), no permanent change in the tympanic membrane arises as a result of otitis.

No difference in the size of the air cell system related to presence or absence of discharge could be established in the present investigation for either marginal or central perforation. I have discussed the question of the nomenclature of chronic otitis, and on the basis of the literature cited I maintain that the nomenclature is inadequate as referring to the processes of disease under consideration. In spite of this I have adopted the definition given by Brock in the textbook of Denker and Kahler. The inadequacy of the nomenclature is not important for the present exposition, as the changes of the tympanic membrane brought together in the different groups have been clearly defined.

It may finally be pointed out that the classification was not based on the presence of cholesteatoma. I disregarded this on account of the factors of inaccuracy relating to the establishment of a diagnosis of cholesteatoma. This tumor may be found in a number of cases, but in those in which it cannot be diagnosed, one cannot with any certainty exclude its occurrence. It may be so small that it can be demonstrated only microscopically. Besides, it may be present in an ear with central perforation of the tympanic membrane without being demonstrable in an otoscopic examination. Finally, it may be present behind an entirely normal tympanic membrane. Certain factors seem indirectly to indicate

that it is intimately connected with a certain size of the cell system, but no definite statement can be made.

Examination of the patients with acute otitis shows that this disease develops in ears with cell systems which, on an average, are smaller than those of normal subjects. Both in the age group under 10 years and in that over 10 years the average values for acute otitis show statistically significant differences as compared with the values for the normal subjects in the corresponding age groups. In the age group 10 to 30 years this difference amounts to about 20 per cent. This means that persons with acute otitis have on an average four fifths as large air cell systems as normal persons. Persons with the largest cell systems, corresponding to the values exceeding 23 sq. cm., practically in no circumstances contract ordinary acute otitis. As compared with the cases of chronic otitis, the cases of acute otitis present a statistically significant higher average value.

The table gives a survey of this part of my investigation.

Summary of Data

Diagnosis	Ears Examined	Age Group, Years	Average Value of Cell System, Sq. Cm.	Standard Error, Sq. Cm.	Highest Value of Cell System, Sq. Cm.
Normal material	356	10-30	12.27	±0.40	27.6
Chronic otitis	138	10-30	2.89	±0.27	15.1
Marginal perforation	101	10 and over	1.68	±0.21	9.2
Central perforation	136	10 and over	3.54	±0.30	15.1
Atrophic scar	78	10 and over	4.04	±0.46	16.4
Acute otitis	91	10 and over	9.60	±0.48	20.5

I wish to emphasize that the conception of chronic otitis in accordance with Brock's definition refers to the state of the tympanic membrane and not to the type of infection. Thus, if a person contracts acute otitis and has symptoms characteristic of this disease and if the roentgenogram shows a small cell system or even entire absence of air cells, one has to reckon with the possibility that the final result of the acute otitis in question may be the origin of a permanent change in the tympanic membrane (central perforation of the membrane, i.e., "chronic otitis" possibly followed by atrophic scar formation of the membrane). However, one has no right or reason to assume that acute otitis in cases in which there is moderate development of the cell system will take a clinical course with regard to uncomplicated healing or to complications from involvement of the labyrinth, sinus, meninges and other structures differing from that of acute otitis in an abundantly pneumatized ear. One may maintain that otitis in a patient in whom pneumatization of the temporal bone has been wholly inhibited cannot

result in mastoiditis, as cell systems are lacking. But an operation corresponding to "chiseling" may well be indicated by the aforementioned, or other, complications.

A special group for investigation was made up of cases of scarlatinal otitis. It was shown that this disease affects the smaller cell systems in persons under the age of 10 years. For the age group over 10 years such a predilection could not be demonstrated in the investigation reported here. It is, however, possible that an examination based on a larger series might show that the given rule applies also to the age group over 10 years. My series of cases is not sufficiently large to give definite information on this point.

Examination of the normal material finally shows that a far from inconsiderable asymmetry is present between the sizes of the cell systems of the two temporal bones in the same person. But it is considerably smaller than the total variability in the material. This means that there exists a correlation between the sizes of the two cell systems in the same person. (This correlation was determined in figures, and it amounts to $r = 0.86 \pm 0.025$.) In other words, in the normal person the cell systems may be of different sizes, but if one is very large the other is also as a rule fairly large, and conversely.

It is evident that the asymmetry is of great interest. If there exists a connection between small cell systems and a tendency to have otitis, otitis may well be unilateral, even if this correlation is marked.

It was pointed out that in the normal person the sizes of the two cell systems show a correlation. A corresponding correlation is also present in persons affected by chronic otitis. Thus, in persons with unilateral chronic otitis, who consequently have small cell systems in their diseased ears, the cell systems of the healthy ears present an average value falling short of that of the normal material. However, the value considerably exceeds that of the cell systems of ears with chronic otitis. The average difference (asymmetry) between the two ears therefore is greater in persons with unilateral chronic otitis than in normal persons. This is best explained in the following manner: Chronic otitis has a marked correlation to small cell systems. If a person has large cell systems in both ears, chronic otitis will not develop in either of them. If he has small cell systems in both ears, he runs a certain risk of contracting chronic otitis on both sides. If, on the other hand, a person has one large and one small cell system, he runs this risk only in regard to the ear with the small cell system, and consequently unilateral otitis may develop. The investigation also shows that in cases of chronic unilateral otitis in which there is appreciable asymmetry, the smaller cell system is invariably the one affected.

The matter may also be viewed from this angle: The person with a high degree of asymmetry runs a risk of contracting chronic unilateral otitis, since one ear has a small cell system. The marked correlation between chronic otitis and small cell systems also manifests itself in the fact that persons with chronic bilateral otitis have only slight asymmetry with a lower average difference between the two ears than is found in the normal material.

Likewise, in unilateral acute otitis, if there is a marked difference in the size of the two cell systems, it is the ear with the smaller system that is affected. If a small difference is present, the larger cell system may become the site of acute otitis, although this happens less frequently.

This investigation thus supports the current view on the correlation between the size of the air cell system and chronic otitis, and the statistical analysis gives a clearer picture of the situation. The correlation is in certain respects surprisingly strong and considerably more pronounced than could formerly be established. The current view with regard to acute otitis has proved to be incorrect. Even in the case of this disease the risk run by persons with small cell systems is greater than that run by persons with large cell systems.

One may ask, "What risk is run by a person who has air cell systems of a certain size?" Direct figures apply only to a certain population group, i. e., the very group subjected to the examination in question. This is due to the fact that conditions, e. g., the spread of infections, may vary within different parts of a country or in different countries. (It is here assumed that infection is the cause of otitis, although, as has already been pointed out, this is doubtful with regard to the chronic otitis with marginal perforation.) But the relation between frequency figures for the sizes of different air cell systems is probably of greater interest. Possibly persons whose air cell systems are of a certain size run twice the risk of contracting chronic otitis that persons whose air cell systems are of a certain other size do. To what extent small air cell systems predispose to chronic otitis, however, this investigation cannot show. The material did not allow of a direct frequency calculation. The investigation, on the other hand, shows that there are persons with very small air cell systems who escape otitis. These points of view have been specially stressed because I want to warn against an overestimation of the results obtained. The investigation has proved only that persons with small air cell systems run particularly great risks. If a person is discovered on examination to have small air cell systems, it should be remembered that this is not to be disregarded, and if the case is difficult to diagnose, the size of the air cell system may possibly be decisive. But one ought not to base a diagnosis solely on the size of the air cell system.

Finally, it should perhaps be pointed out that, although the size of the air cell system has proved to be an important factor in the development and course of otitis, the more detailed mechanism is not known. Wittmaack's views on the significance of the type of mucous membrane have not been examined by me. As I have been able to arrive only at general conclusions, not at quantitative estimates of frequency, it is evident that hypotheses which are based on the establishment of possible types of mucous membrane by registration of the size of the air cell system in the roentgenogram must for the present be considered incapable of yielding a definite solution of the problems.

I have touched on the problem of hereditary factors but my material does not permit elucidation of this question. If, however, the size of the air cell system is hereditarily determined and the established state of asymmetry does not contradict such an assumption, hereditary factors seem to be of a certain importance for the development of both chronic and acute otitis.

RHINITIS: A NASOGRAPHIC STUDY

PRELIMINARY REPORT

LOUIS E. SILCOX, M.D.
PHILADELPHIA

A simple accurate method for the quantitative study of nasal patency has long been desired by clinicians and rhinologic research workers. The idea of breathing through the nasal passages directly onto a previously cooled mirror or metal surface was introduced in 1889 by Zwaardemaker.[1] He later reported the use of his metal mirror as an aid in the diagnosis of nasal obstruction. In 1895 Kayser,[2] after using the same technic, published his work on "the exact measurement of the nasal air passages." These measurements were derived from the size and shape taken by the moisture deposited on the metal mirror. In 1904 Glatzel[3] modified the mirror by adding two curved lines to aid in determining the degree of nasal patency. In 1915 Cocks[4] added some straight lines to this modified mirror and attempted to measure more accurately nasal conditions before and after operations by the conformity of the moisture deposits on the mirror to the lines. This same investigator did extensive work on the changes in the nasal airways under various atmospheric conditions.[5] No mention of the quantitative relationships in percentile values was made until Mulinos and Lieb[6] recently measured the time of evaporation of the moisture deposits on the mirror under controlled conditions and made a percentage comparison of the two nasal cavities. Other methods of measuring air

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From the Departments of Otolaryngology and Allergy, Hospital of the University of Pennsylvania.

1. Zwaardemaker, H.: Athembeschlag als Hülfsmittel zur Diagnose der nasalen Stenose, Arch. f. Laryng. u. Rhin. **1**:176, 1893.

2. Kayser, R.: Die exacte Messung der Luftdurchgängigkeit der Nase, Arch. f. Laryng. u. Rhin. **3**:101, 1895.

3. Glatzel: Zur Prüfung der Luftdurchgängigkeit der Nase, Monatschr. f. Ohrenh. **38**:8, 1904.

4. Cocks, G. H.: Improved Glatzel Mirror, Laryngoscope **25**:135, 1915.

5. Cocks, G. H.: Experimental Studies of the Effect of Various Atmospheric Conditions upon the Upper Respiratory Tract, Laryngoscope **25**:603, 1915.

6. Mulinos, M. G., and Lieb, C. C.: Nasograph Mirror of Glatzel as a Measure of Nasal Patency, Arch. Otolaryng. **30**:334 (Sept.) 1939.

breathed through the nose have been introduced by various authors,[7] but most of these entail complicated apparatus, which makes the procedures impracticable. A personal communication from Prof. W. Brünings, in Munich, Germany, indicates that the mirror principle is widely used in that country in routine practice but that scientific data are scant.

OBJECTIVES

This work was undertaken with several problems in view. The intentions were:

1. To test the efficiency of the nasograph in a quantitative study of rhinitis.

2. To observe the effect of a solution of a vasoconstrictor (paredrine hydrobromide aqueous) in various forms of rhinitis.

3. To compare quantitatively the vasoconstrictor effect of two different concentrations of the solution in different pathologic conditions of the nasal mucous membranes.

4. To observe the effect of such a solution applied locally to allergic mucous polyps.

5. To investigate a new approach for estimating the effectiveness of injection therapy in ragweed pollinosis.

DESCRIPTION OF THE NASOGRAPH

The nasograph mirror consists of a rectangular piece of polished stainless steel, one of the long sides of which has been cut to conform to the convexity of the upper lip. At right angles to this cut is placed a removable bow-shaped metal strip (1 cm. in height), notched to accommodate the columella. The opposite long edge is slotted and provided with a thumb screw fitting for easy attachment (at right angles) to the holder. The under side of the mirror is provided with a shallow water reservoir, to maintain a constant temperature of the mirror of 25 C. for a maximum of five minutes. When not in use, the mirror is placed in water which is maintained at a constant temperature of 25 C.

The holder for the nasograph mirror consists of a straight steel bar with a wooden hand grip at the bottom and a hole through the top to accommodate an adjustable brow rest at right angles to it. Along the length of the bar is an adjustable slide to receive the mirror described.

The nasograph (the mirror and mirror holder assembled) is fitted to the patient in such a way that the metal strip of the mirror is centrally located under the orifices of his nose and the mirror itself at right angles to the frontal plane of his face, the latter position being effected by means of the adjustable brow rest.

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7. Sternstein, H. J., and Schur, M. O.: Quantitative Study of Nasal Obstruction: A New Method, Arch. Otolaryng. **23**:475 (April) 1936.

CLASSIFICATION OF SUBJECTS

Of 112 patients observed and tested, 35 were found to be unsuitable for study, for reasons which will be discussed later. Of the 77 patients studied, 65 had allergic rhinitis and 12 had rhinitis due to other causes.

The patients with allergic rhinitis were classified as follows:

(*A*) Those with complete nasal occlusion due to contact with airborne ragweed pollen.

(*B*) Those with partial nasal occlusion due to contact with airborne ragweed pollen.

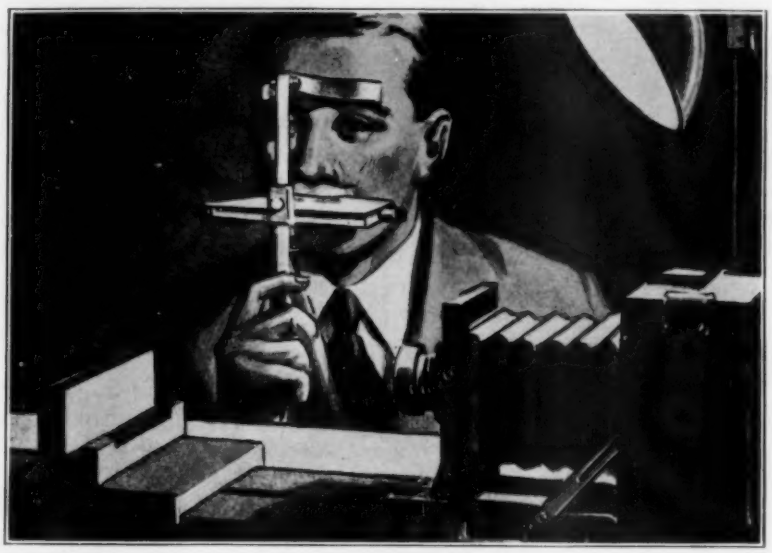

Fig. 1.—Nasograph in use.

(*C*) Those with complete nasal occlusion induced by the direct application of an extract of ragweed pollen to the nasal mucous membranes.

(*D*) Those with partial nasal occlusion induced by the direct application of the ragweed pollen extract to the nasal mucous membranes.

The patients with rhinitis of nonallergic causation were classified as follows:

(*A*) Those with complete occlusion from acute rhinitis.

(*B*) Those with partial occlusion from acute rhinitis.

(*C*) Those with partial occlusion from chronic rhinitis.

Any observer working with the problem of nasal patency on a quantitative basis must take certain variables into consideration. Those

found to be important were: changes in room temperature or humidity; normal physiologic shifting or change in the size of the turbinates; fluid or secretions in the nose, allergic edema fluid, pus, nose drops or any combination of these; septal deflections with or without ridges and spurs; multiple or single large polyps; chronic hypertrophy or hyperplasia of the turbinates, and the state of the allergic patient's sensitivity, whether influenced by treatment, spontaneous or resulting from an induced attack of allergic rhinitis. A patient who inadvertently "sniffs" can change the whole quantitative relationship of one nasal airway to the other.

There were certain limitations of the nasograph mirror, and many of the patients observed and tested were not studied further because of one or more of these factors. Inasmuch as this work is entirely a comparison of one side of the nose with the other, it is obvious that if the deposits of moisture on the mirror do not split into two separate and distinct components, there can be no comparison. This occurred in a few instances. In patients whose less patent nasal airway showed over 35 per cent of the total, considering the total moisture from both nostrils as 100 per cent, it was found that there was relatively less shrinking to do, and the vasoconstrictor studies on these patients were of less value.

METHOD

The vasoconstrictor used was paredrine hydrobromide aqueous.[8]

The ragweed pollen extract, dilution 1:53, used for the induction of allergic rhinitis in some of the patients, was prepared at the allergy clinic of the Hospital of the University of Pennsylvania.

The patient to be tested was seated and told to breathe as naturally as possible and not to blow his nose. When complete unilateral or bilateral occlusion made breathing difficult, instructions were given to inhale through the mouth and to try to exhale through the nose.

The patient's nose was examined and all salient information recorded.

The nasograph was applied during one of the patient's inhalations, and the moisture from three exhalations was permitted to condense on the face of the mirror, after which the instrument was removed. At the exact moment of removal two stopwatches were started—unless there was complete unilateral occlusion, when only one was used.

The moisture spot or spots were observed closely, and when each disappeared, the corresponding watch was stopped. The duration of each spot was recorded in seconds, and the shorter time was considered a measure of the less patent

8. Paredrine hydrobromide aqueous is a synthetic compound (*p*-hydroxy-*α*-methylphenethylamine hydrobromide; hydroxyamphetamine) in an isotonic solution, buffered with sodium borate to p_H 6.8 and preserved with merthiolate, 1:100,000. It was supplied by the Smith, Kline & French Laboratories, Philadelphia.

nostril. Using the symbol OL for the duration of the spot made by the left nostril and OR for that of the one made by the right, the percentile value of patency in the more occluded nostril was calculated by means of this formula:

$$\frac{\text{OR or OL (whichever is less patent)}}{\text{OR} + \text{OL}} \times 100.$$

The foregoing procedures were repeated at intervals of from one to three minutes, to check the percentile relationship. A relatively constant percentile patency (± 5 per cent) was sought before proceeding.

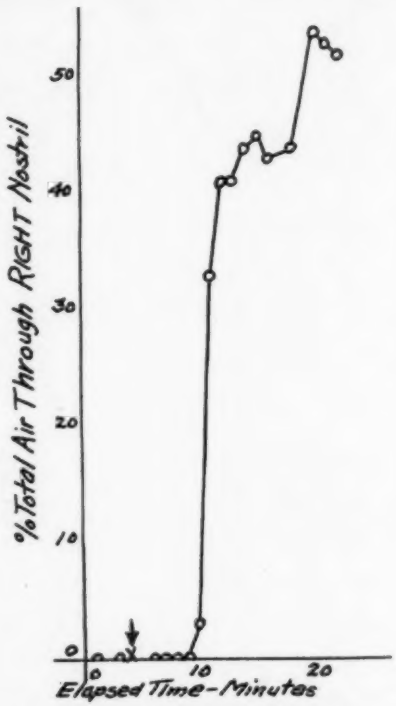

Fig. 2.—Effect of a vasoconstrictor (1 per cent paredrine hydrobromide aqueous) on a patient with acute rhinitis with complete unilateral nasal occlusion. *X* indicates the beginning of treatment.

When a reasonably stable nasal condition was reached, one of two procedures was followed:

1. During the ragweed pollen season ragweed-sensitive patients presented themselves with either partially or completely occluded nostrils. In patients with partially occluded nostrils, an attempt was made to render one nostril completely closed by instilling ragweed pollen extract (1:53) directly on an inferior turbinate.

All reactions were noted during a lapse of ten minutes, after which the mirror was reapplied, to determine the status of nasal patency, check readings being taken until stability was again reached. The nose was reexamined and the findings recorded. In those cases in which complete occlusion was caused or one

nostril became even less patent than before, either 1 or 3 per cent paredrine hydrobromide aqueous was administered. If, to begin with, both nostrils had been nearly even and no reduction in patency had occurred with the treatment, this fact was recorded and the patient was discharged.

Those patients with at least one completely occluded nostril were treated immediately with either 1 or 3 per cent paredrine hydrobromide aqueous.

2. When ragweed-sensitive patients were no longer seen in the clinics, other types of rhinitis were studied, and these were treated simply with paredrine

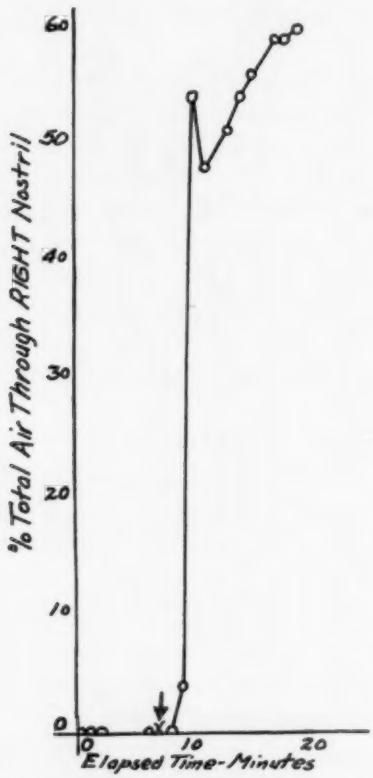

Fig. 3.—Effect of a vasoconstrictor (3 per cent paredrine hydrobromide aqueous) on a patient with acute rhinitis with complete unilateral nasal occlusion. *X* indicates the beginning of treatment.

hydrobromide aqueous after nasographic readings showed the patency of either side to be consistently less than 35 per cent of the total.

Following the instillation of paredrine hydrobromide aqueous, nasographic readings were taken at one minute intervals for at least twelve minutes. The treated nostril was examined several times during this period and after the last reading had been taken, all observations being recorded as made. At this point, unless interesting information appeared to be forthcoming, the patient was discharged.

Readings of room temperature and relative humidity were taken and recorded either during or immediately after a test.

From the data obtained by the foregoing procedures, curves for individual patients were plotted, values for elapsed time being used as abscissas and percentile values for total air passed through either the left or the right nostril (whichever was the less patent originally) as ordinates.

From the curves, a value of optimum reaction time was obtained for a given patient by following the course of nasal patency in the

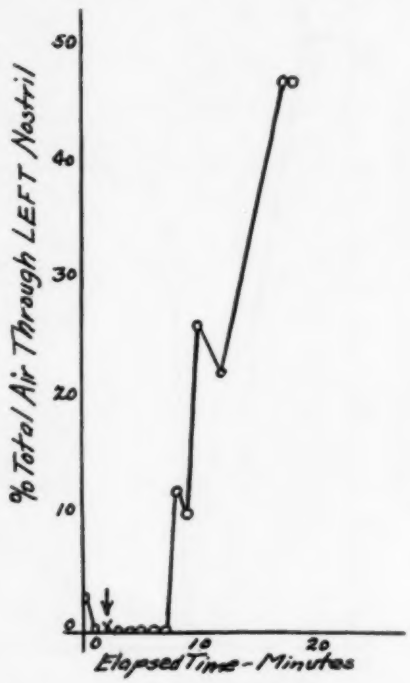

Fig. 4.—Effect of a vasoconstrictor (1 per cent paredrine hydrobromide aqueous) on a patient with allergic rhinitis with spontaneous complete unilateral nasal occlusion. *X* indicates the beginning of treatment.

treated nostril until a point was reached that showed a minimum of 10 per cent improvement and also abatement in the rate of mucosal shrinkage. In tables 1 to 4 the optimum reaction time and the improvement in patency are given for each patient studied.

Improvement in patency, as shown, is the difference between patency at the time of instillation of the drug and that at optimum reaction time.

An analysis of the graphic data indicated case types; common characteristics suggested a grouping of cases as in tables 1 to 7.

Acute or chronic rhinitis, including that due to allergy, with complete occlusion (table 1).

Acute or chronic rhinitis, including that due to allergy, with partial occlusion (table 2).

Allergic rhinitis induced with ragweed pollen extract, resulting in complete occlusion (table 3).

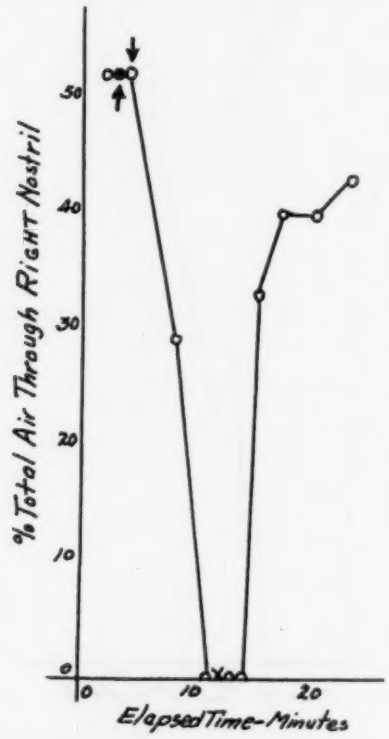

Fig. 5.—Effect of a vasoconstrictor on a patient with allergic rhinitis induced by intranasal instillation of ragweed pollen extract (indicated by the black dot) and producing complete unilateral occlusion. The white dot indicates the times the patient sneezed. *X* indicates the beginning of the administration of paredrine hydrobromide aqueous.

Allergic rhinitis induced in the same way, resulting in partial occlusion (table 4).

Summary of the averages taken from tables 1 to 4 and grand averages of optimum reaction time and improvement in patency for all reactive

TABLE 1.—*Acute or Chronic Rhinitis, Including That Due to Allergy, with Complete Occlusion*

Patient	1% Paredrine Hydrobromide Aqueous		3% Paredrine Hydrobromide Aqueous	
	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent
O. T.	7	22	..	..
D. A.	3	100	..	..
L. P.	8	26	..	..
E. C.	5	21	..	..
J. R. (polyposis)	6	48	..	..
A. H.	7	19	3	54
F. S.	15	29	5	100
L. H.	5	70	4	65
P. L.	..	..	8	30
W. B.	..	..	7	40
L. L.	..	..	4	54
P. B.	..	..	6	100
M. G. (polyposis)	..	..	5	76
Average	7	42	5	66

TABLE 2.—*Acute or Chronic Rhinitis, Including That Due to Allergy, with Partial Occlusion*

Patient	1% Paredrine Hydrobromide Aqueous		3% Paredrine Hydrobromide Aqueous	
	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent
A. M. (polyposis)	6	23	..	..
L. W.	1	11	..	..
P. L.	4	18	..	..
M. B.	14	41	..	..
J. D.	1	18	..	..
D. L.	7	19	..	..
M. K.	3	20	5	14
M. F.	3	15	3	13
M. M.	3	31	5	37
J. F.	3	27	5	23
Y. C.	4	14	4	18
R. D.	..	..	8	21
O. T.	..	..	3	16
S. J.	..	..	10	25
L. P.	..	..	4	12
I. C.	..	..	3	11
F. Y.	..	..	7	26
M. Ma.	..	..	4	22
E. W.	..	..	7	39
G. F.	..	..	4	15
Average	4	22	5	21

TABLE 3.—*Allergic Rhinitis Induced with Ragweed Pollen Extract Resulting in Complete Occlusion*

Patient	1% Paredrine Hydrobromide Aqueous		3% Paredrine Hydrobromide Aqueous	
	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent
B. R.	11	31	..	..
M. F.	2	41	3	33
J. D.	..	..	4	29
R. M.	..	..	9	44
Average	7	36	5	35

patients studied, comparing the effectiveness of 1 per cent and 3 per cent paredrine hydrobromide aqueous in general (table 5).

All types of rhinitis: unstable, unreactive to allergen and/or paredrine hydrobromide aqueous, with the reason for condition observed (table 6).

TABLE 4.—*Allergic Rhinitis Induced with Ragweed Pollen Extract Resulting in Partial Occlusion*

	1% Paredrine Hydrobromide Aqueous		3% Paredrine Hydrobromide Aqueous	
Patient	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent
R. J.	4	14	..	..
A. P.	11	16	..	..
M. L.	8	12	..	..
M. P.	3	9	..	..
E. A.	8	16	..	..
M. K.	5	10	..	..
L. L.	6	23	..	..
E. M.	6	15	3	28
B. R.	..	..	9	13
Average	6	14	6	21

TABLE 5.—*Summary of the Averages Taken from Tables 1 to 4 and Grand Averages of Optimum Reaction Time and Improvement in Patency for All Reactive Patients Studied, Showing the Comparative Effectiveness of 1 per Cent and 3 per Cent Paredrine Hydrobromide Aqueous*

	1% Paredrine Hydrobromide Aqueous		3% Paredrine Hydrobromide Aqueous	
	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent	Optimum Reaction Time, Minutes	Improvement in Patency, per Cent
Table 1: Acute and chronic rhinitis, with complete occlusion	7	42	5	66
Table 2: Acute and chronic rhinitis, with partial occlusion	4	22	5	21
Table 3: Allergic rhinitis, induced attack with complete occlusion	7	36	5	35
Table 4: Allergic rhinitis, induced attack with partial occlusion	6	14	6	21
Grand average	6	26	5	36

Chronologic arrangement of reactive patients with respect to optimum reaction time (table 7).

If all the patients studied (regardless of the aforementioned classification) are arranged according to their respective optimum reaction time (table 7), by noting the improvement in patency for each, it is possible to develop two curves showing (fig. 6) the averge improve-

ment in patency and (fig. 7) the number of patients reacting plotted against increasing values of optimum reaction time.

It is logical to assume that if the nasograph is of value in measuring the action of vasoconstrictors in terms of the increase in nasal patency, then the converse should be true, and it should be possible to measure the exact degree of reactivity of the nasal mucous membranes to sensitizing agents in terms of decrease in nasal patency.

When pollen-sensitive patients are inoculated preseasonally with increasing doses and strengths of pollen extract, the effect of the desensi-

TABLE 6.—*All Types of Rhinitis: Unstable, Unreactive to Allergen and/or Paredrine Hydrobromide Aqueous, with the Reason for Condition Observed*

	Patient	Condition Observed
Allergic rhinitis	H. K.	Complete occlusion due to polyposis
	S. M.	Complete occlusion due to polyposis
	R. S.	Complete occlusion due to polyposis
	M. G.	Complete occlusion due to polyposis
	M. Go.	Attack induced with ragweed extract, so intense that vasoconstrictor failed to shrink the mucosa
	J. C.	Attack induced with ragweed extract, so intense that vasoconstrictor failed to shrink the mucosa
	D. A.	Complete occlusion from acute allergic edema
	M. O.	Complete occlusion from acute allergic edema
	A. M.	Complete occlusion due to excess fluid
	E. B.	Partial occlusion with patency above 35 per cent
	M. B.	Partial occlusion with patency above 35 per cent
	L. W.	Unreactive to allergen; receiving allergic therapy; unreactive to vasoconstrictor; patency above 35 per cent
	G. D.	Unreactive to allergen; receiving allergic therapy; unreactive to vasoconstrictor; patency above 35 per cent
	E. L.	Unreactive to allergen; receiving allergic therapy; unreactive to vasoconstrictor; patency above 35 per cent
	C. W.	Chronic hypertrophy of left inferior turbinate
	S. B.	Bilateral inferior turbinectomy
	J. R.	Unstable; septum extremely deviated
Acute rhinitis	I. S.	Partial occlusion with patency above 35 per cent
	G. C.	Partial occlusion with patency above 35 per cent
Chronic rhinitis	R. Y.	Unstable; septum extremely deviated
	E. J.	Unstable; septum extremely deviated

tization is to render the mucous membranes of the upper respiratory tract more resistant to the irritating properties of that pollen. All rhinologists are familiar with the typical picture of ragweed pollinosis at the start of the hay fever season in a patient who has not received preseasonal injections.

A test to determine sensitivity to a specific pollen is to apply that pollen or an extract of it directly on the nasal mucous membranes. If there are sneezing, swelling and other signs or symptoms of reaction, it is known that the person is not protected against that pollen.

Twenty-five patients with sensitivity to ragweed pollen were studied in the following manner: 1 drop of ragweed pollen extract, dilution

1 : 53, was placed directly on the mucous membrane in the less patent nasal cavity, and nasographic readings were made of the change in percentile relationship between the side treated with the extract and the untreated side.

TABLE 7.—*Chronologic Arrangement of Reacting Patients with Respect to Optimum Reaction Time*

1% Paredrine Hydrobromide Aqueous				3% Paredrine Hydrobromide Aqueous			
Optimum Reaction Time, Minutes	Percentage of Total Patients	Improvement in Patency, per Cent	Average per Cent	Optimum Reaction Time, Minutes	Percentage of Total Patients	Improvement in Patency, per Cent	Average per Cent
1		11		3		54	
1	6.9	18	15	3		28	
				3		13	
2	3.4	41	41	3		16	
				3		11	
3		100		3	22.2	33	26
3		20					
3		15		4		29	
3		31		4		18	
3		27		4		12	
3	20.7	9	34	4		22	
				4		15	
4		14		4		65	
4		18		4	25.9	54	31
4	10.4	14	15				
				5		100	
5		10		5		76	
5		70		5		14	
5	10.4	21	34	5		37	
				5	18.5	23	50
Total	51.8	Average	28	Total	66.6	Average	36
6		48		6	3.7	100	100
6		23					
6		23		7		40	
6	13.8	15	27	7		26	
				7	11.1	39	35
7		22					
7		19		8		21	
7	10.4	19	20	8	7.4	30	26
8		26					
8		12		9		44	
8	10.4	16	18	9	7.4	13	29
11		16					
11	6.9	31	24				
14	3.4	41	41				
15	3.4	29	29	10	3.7	25	25
Total patients 29				Total patients 27			

Twenty of these patients had received preseasonal injections for one or more years previous to the testing; the other 5 had received no treatments.

Of the 20 treated patients, 4 reacted to the intranasal application of the extract, showing a change in nasal patency from as much as 45 per cent to complete occlusion in from five to twenty minutes.

Of the 5 untreated patients, 4 reacted to the extract, showing a change in nasal patency from as much as 50 per cent to complete occlusion in from eight to seventeen minutes. Some of the patients in each

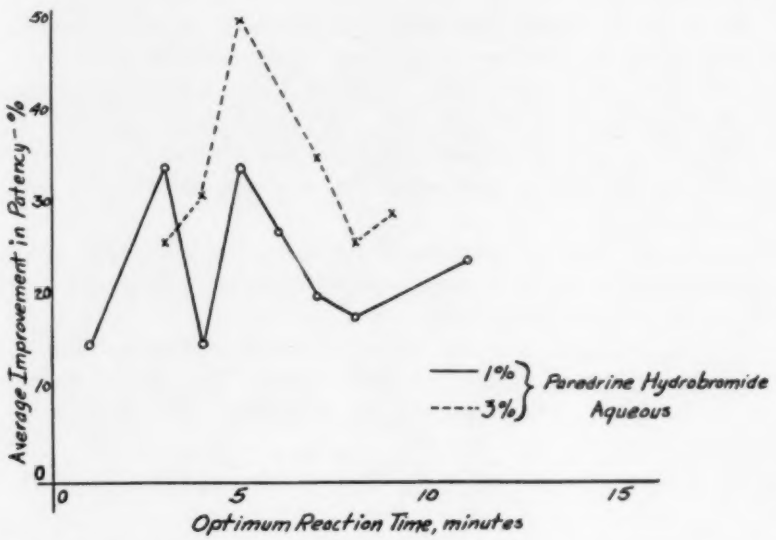

Fig. 6.—Comparative curves, showing improvement in patency for various values of optimum reaction time.

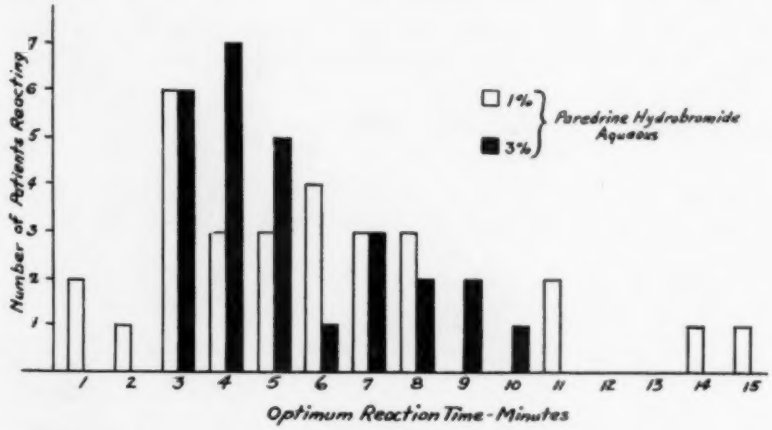

Fig. 7.—Number of patients reacting at various values of optimum reaction time.

group had repeated examinations on successive days, for checking, and the general response was found to be the same in each case.

SUMMARY

The modified nasograph mirror, as described in the text, was found to be an efficient clinical instrument for the quantitative study of rhinitis of all forms.

The solution tested (paredrine hydrobromide aqueous) produced effective vasoconstriction in 73 per cent of the patients studied.

The average optimum reaction time for 1 per cent paredrine hydrobromide aqueous is six minutes, with an improvement in patency of 26 per cent. The average optimum reaction time for the 3 per cent concentration of the drug is five minutes, with an improvement in patency of 36 per cent.

In patients with complete nasal occlusion due to allergic mucous polyps, paredrine hydrobromide aqueous in the concentration used showed no vasoconstrictor effect.

The observations made on patients in whom allergic rhinitis had been induced suggest that controlled induction of ragweed pollinosis may be used as a new approach for estimating the effectiveness of injection therapy in that condition.

SENILE CHANGES IN THE LARYNGEAL MUSCULATURE

AMIL C. BACH, M.D.
INDIANAPOLIS
AND
FRANCIS L. LEDERER, M.D.
AND
ROBERT DINOLT, M.D.
CHICAGO

The larynx of old persons has frequently been examined. However, a perusal of the literature reveals that no two physicians agree in their findings as far as the muscles of this organ are concerned. Kofler [1] mentioned slight atrophy of the muscles, without giving further details, and Imhofer [2] found that the lipoid pigment usually situated around the nuclei of the sarcolemma increased in amount in old persons. Segre [3] found simple atrophy of the adductor and the abductor muscles, characterized by an increase of the nuclei of the sarcolemma and an increase in both the collagenous and the elastic fibers. This atrophy was least marked in the interarytenoid and the vocal muscles. In contrast, Imhofer [4] did not observe an increase in elastic fibers. The cricothyroid muscles showed hypertrophy in the cases of Segre,[3] which was considered compensatory, because of the atrophy of the adductor muscles. Carnevale Ricci [5] described essentially the same conditions as Segre [3] except for the lack of compensatory hypertrophy of the cricothyroid muscles and, in addition, observed fatty degeneration of the laryngeal muscles.

Because the changes found in the laryngeal muscles are not sufficient to explain the gross changes of the senile larynx and the characteristic

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From the Department of Pathology of the Cook County Hospital and the Department of Otology, Rhinology and Laryngology of the University of Illinois College of Medicine.

1. Kofler, K.: Die Altersveränderungen im Larynx, Monatschr. f. Ohrenh. **66**:1468, 1932.

2. Imhofer, F.: Ueber das Abnutzungspigment in der Muskulatur der Stimmbänder, Ztschr. f. Laryng., Rhin. **5**:389, 1912.

3. Segre, R.: La laringe senile, Valsalva **14**:15, 1936.

4. Imhofer, F.: Ueber das elastische Gewebe im Stimmband alter Individuen, Zentralbl. f. allg. Path. u. path. Anat. **25**:337, 1914.

5. Carnevale Ricci, F.: Osservazioni istopatologiche sulla laringe nella senescenza, Arch. ital. di otol. **49**:1, 1937.

changes of the senile voice and because no attempt has been made to correlate the alterations in the larynx with the disease which led to the death of the patient, we decided to examine further the muscles of the senile larynx. Serial sections of laryngeal muscles from 5 well nourished patients between 60 and 80 years of age, with no previous laryngeal complaints, were made and stained with hemalum and eosin; frozen sections were stained with sudan. The muscles were divided into three groups: the adductor, the abductor, or posterior cricoarytenoid, and the tensor, or cricothyroid, muscles.

CASES IN WHICH MICROSCOPIC EXAMINATION OF THE LARYNGEAL MUSCLES WAS MADE

CASE 1.—The patient was a colored man aged 62. A clinical diagnosis was not made. The anatomic diagnosis was: large chronic duodenal ulcer, hemorrhagic gastritis, saccular aneurysm of the aorta, syphilitic aortitis and obliterative pericarditis.

CASE 2.—The patient was a white man aged 79. The clinical findings were: moderate obesity, slight sore throat a few days before admission, injection of the pharynx, marked postnasal drip and painful swelling of the joints of two months' duration. The anatomic diagnosis was: moderate hypertrophy of the heart, moderate coronary sclerosis, generalized arteriosclerosis and chronic arthritis.

CASE 3.—The patient was a white man aged 66. The clinical impression was that of Hodgkin's disease, obstructive dyspnea and dysphagia. The anatomic diagnosis was: generalized retothel sarcoma of the mediastinal, cervical, axillary, peribronchial, periaortic, inguinal and mesenteric lymph nodes, compression of the trachea and the lower bronchi by a tumor and metastases to the left parotid gland, the wall of the stomach and the left pleura.

Case 4.—The patient was a white man aged 63. The clinical diagnosis was: coronary thrombosis and chronic mastoiditis and otitis media on the right side. The Kahn and the Wassermann test were positive. The anatomic diagnosis was: ulcerating adenocarcinoma of the stomach, syphilitic aortitis, slight periportal cirrhosis, chronic otitis media on the right side and syphilitic leptomeningitis.

CASE 5.—The patient was a white man aged 72. The clinical diagnosis was: lobar pneumonia of the right lung. The anatomic diagnosis was: extensive focal and confluent bronchial pneumonia and moderate cardiac hypertrophy.

MICROSCOPIC OBSERVATIONS ON THE LARYNGEAL MUSCLES IN THE CASES LISTED

Fatty Degeneration.—There was a slight fatty infiltration of the cricothyroid muscles in cases 2 and 5 (fig. 1). It must be emphasized that in both cases the patients were over 70 years of age. In the other cases there was neither fatty degeneration nor storage of fat in any of the muscles.

Increase of Connective Tissue and of Nuclei of the Sarcolemma.—This condition was discovered in all the cases, but to a lesser degree

in case 3. In general, it was most pronounced in the abductor, moderate in the tensor and least noticeable in the adductor muscles. The increase of nuclei and connective tissue was not uniform, but islands of increased connective tissue, of increased nuclei or of both were scattered throughout the muscles. The nuclei of the sarcolemma were rich in chromatin and arranged either in groups or in rows (fig. 2 *A*). The connective tissue was of a loose areolar type and rich in nuclei (fig. 2 *B*). No heavy connective tissue suggesting a scar following inflammation was found. There was a slight round cell infiltration of the fat between the muscle fibers in case 2.

Fig. 1.—Fatty degeneration of cricothyroid muscle.

Disturbances of Blood Vessels.—There were no definite anatomic changes in the blood vessels, even though 2 of the patients were syphilitic and 1 had marked generalized arteriosclerosis. There was, however, definite hyperemia of the capillaries in all cases, and sometimes there was edema of the connective tissue around the capillaries, but more important was the finding of capillary hemorrhages. Since most of these hemorrhages consisted of well preserved erythrocytes, it is doubtful whether all of them occurred during life. Only in case 1 was there definite pigment within the connective tissue between the muscle fibers, proving the intravital occurrence of the bleeding. In case 2 we found a large area of hemorrhagic infarction in which necrotic and fragmented muscle fibers were present (fig. 3). This is undoubtedly explained by the marked generalized arteriosclerosis.

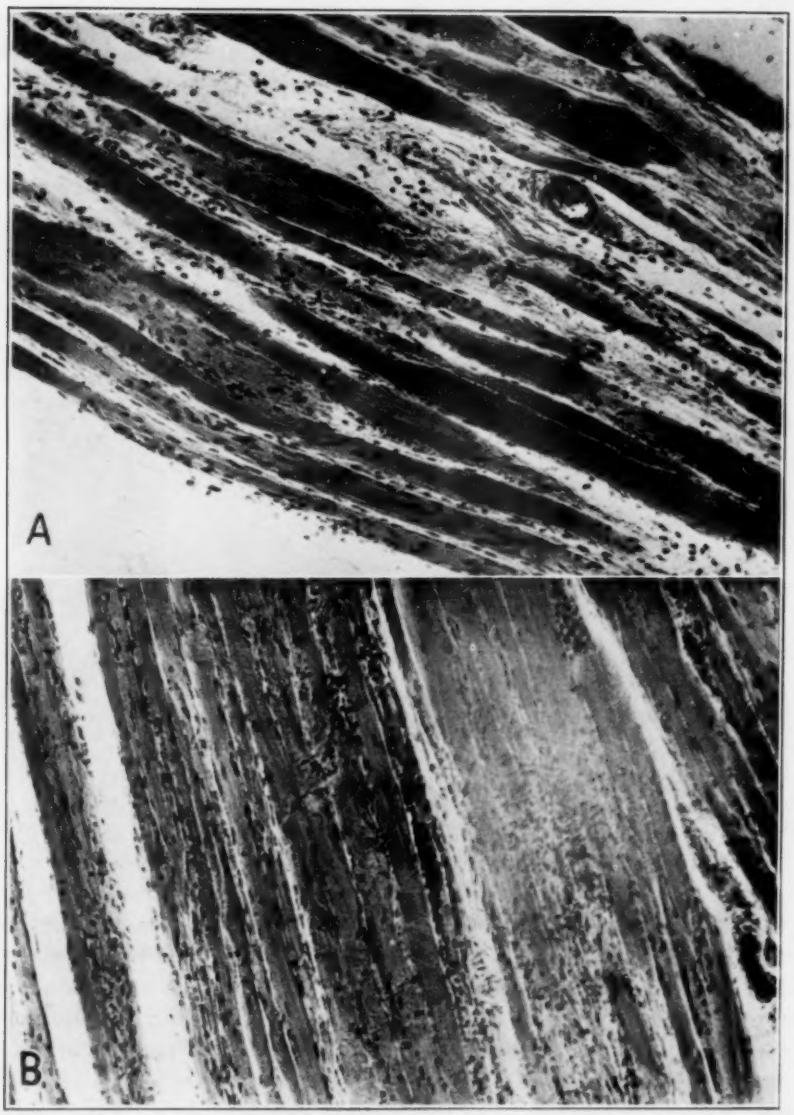

Fig. 2.—*A*, cricothyroid muscle of the patient in case 5, showing varying thickness of fibers, increase of nuclei and partial loss of cross striation. *B*, posterior cricoarytenoid muscle of the patient in case 5, showing increase of connective tissue and of nuclei and empty sarcolemma tubes.

Fig. 3.—Adductor muscle of the patient in case 2, showing a large hemorrhage into the muscle, with tearing of the fibers.

Fig. 4.—Posterior cricoarytenoid muscle of the patient in case 5, showing increase of nuclei and of connective tissue and muscular sequestrums.

Changes in Muscular Tissue.—There were various pathologic changes in the muscle tissue. Frequently a definite difference in the thickness of the fibers was observed. In places the cross striations were absent, yet the longitudinal striations were still present. When these degenerative changes progressed the muscle fibers became entirely homogenous, and many showed a spindle-like swelling, while others showed a "discoid decay," which continued until muscle sequestrums developed (fig. 4). The last step of this degenerative process showed the sheath of the sarcolemma varying from the state of being empty to that of being completely filled with nuclei or remnants of sarcoplasm.

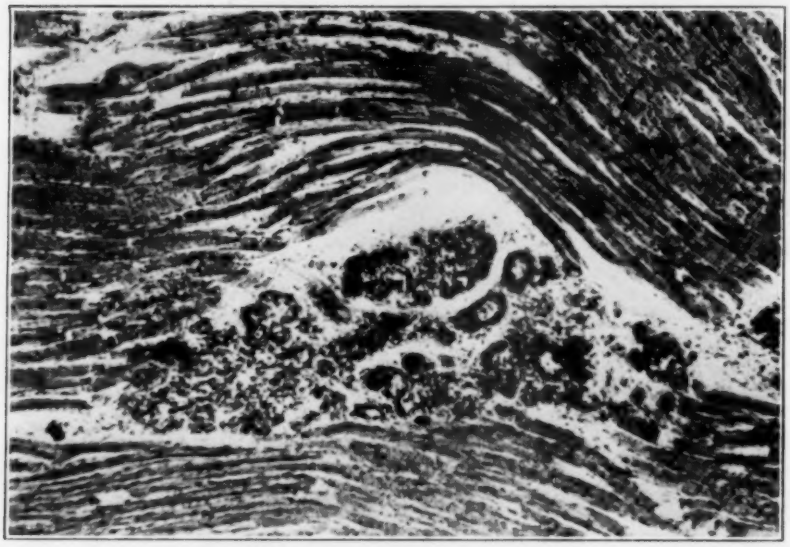

Fig. 5.—Large mucous gland embedded in adductor muscle of the patient in case 3.

The changes described were found in all the muscles examined, the only difference being in their degree. In general, these changes were most marked in the cricoarytenoid muscles (abductors), especially the increase of nuclei and connective tissue.

Glands Within the Muscle.—In case 3 we found a large mucous gland embedded within the adductor muscle (fig. 5). Since the glands are usually found between the epithelial and the muscular layer, this anomaly must be explained as a congenital displacement of glandular tissue.

Tumor Cells Within the Muscle.—Within the abductor muscle in case 3, between the fibers, we found large disklike or polygonal cells with well developed clear protoplasm and small centrally located nuclei.

which were neither connective tissue cells nor cells of the sarcolemma. Since the patient died from a retothelsarcoma, these were considered to be tumor cells, and identification was later confirmed (fig. 6).

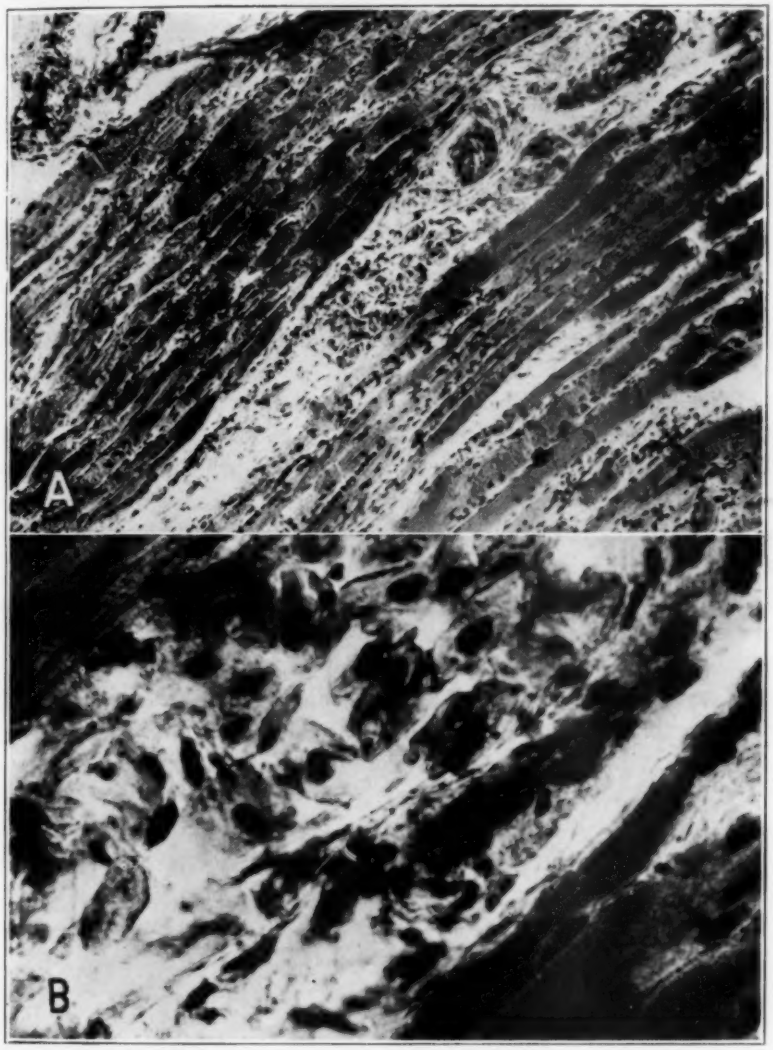

Fig. 6.—*A*, posterior cricoarytenoid muscle of the patient in case 3, showing increase of connective tissue and of nuclei and tumor cells embedded in the interfascicular connective tissue. *B*, tumor cells of *A* under higher power.

COMMENT

After reviewing our findings, we believe that there are definite degenerative changes in the laryngeal muscles of old persons which should be sufficient to explain the peculiarities of the senile voice. We

admit that the number of specimens examined is not large enough to permit us to draw general conclusions. However, we did not intend to determine the frequency of these degenerative changes among old persons but to decide definitely whether or not there were any changes at all. The latter is of practical importance, as one is seldom interested in knowing how many old persons have a senile voice; the important fact is that a senile voice exists. Under these circumstances the question arises as to why these degenerative changes occur; thus, the following points must be considered:

1. The disease which led to the death of the patient.
2. Previous inflammatory diseases of the larynx.
3. Senility.
4. The condition of the blood vessels within the muscles.

It has already been pointed out that pathologic changes of the blood vessels were not found, even in case 2, in which there was marked generalized arteriosclerosis. We also examined specimens from 2 patients with syphilitic aortitis, a condition in which there is frequently inflammatory and mechanical encroachment by the diseased aorta on the inferior laryngeal nerve. This is especially prone to occur when there is an aneurysm, as in case 1. Consequently, one could suppose that the degenerative changes found in cases 1 and 4 were due to syphilitic inflammation of the recurrent laryngeal nerve, even though there was no clinical evidence of paralysis. This explanation, however, does not hold, since under these circumstances the degenerative changes should exist only on the left side and should be entirely absent in the other patients who were not syphilitic. Previous inflammatory conditions of the larynx, which must be considered in the case of every old person, could produce scars within the laryngeal muscles, provided the inflammation invaded the deep layers of the larynx. In our cases there was no evidence of the formation of scars within the muscles. Senility could damage the laryngeal muscles by overaction. That is, however, a rather hypothetic approach to the explanation of the pathologic changes we found. As may be seen later, the same changes we found in the laryngeal muscles which we examined may also be found in those of patients with recurrent laryngeal paralysis. It is difficult to understand how identical pathologic changes are due in one instance to overaction of the muscles and in another to inactivity of the muscles.

Under these circumstances the only possible explanation for the pathologic changes in the laryngeal muscles of old persons is to be found in the vascular disturbances and, consequently, in the insufficient nourishment of the muscles. It is in accordance with this explanation that one meets with the same pathologic conditions in cases of recurrent laryngeal

paralysis. In such a case the findings are not explained by the paralysis of the motor fibers of the recurrent laryngeal nerve (due to inactivity) but by a paralysis of the vasoconstrictor fibers of the sympathetic nerve, the entrance of which into the recurrent laryngeal nerve, through the ansa Galeni, constitutes a vasomotor change. Therefore, we believe that the laryngeal muscles are affected quickly by insufficient nourishment and undergo degenerative changes.

Another obviously important point is in connection with the muscular degeneration which has been frequently demonstrated to be associated with paralysis of the inferior laryngeal nerve. It must necessarily be considered that many of these previously demonstrated changes, especially when they occur in older persons, are not the direct result of the paralysis but are produced by the nutritive changes, as we have demonstrated, independent of the neural involvement. This, of course, holds true only for the minor changes microscopically proved in our series and not for the gross changes of the muscles, which are not found in older persons but are readily observed in patients with paralysis of the recurrent nerve.

In order to prove our point we examined the laryngeal muscles of a 30 year old white man with severe secondary anemia (red blood cells, 1,080,000; hemoglobin content, 20 per cent) due to subacute glomerulonephritis. The changes in his laryngeal muscles were the same as those found in an old person, so there can be no doubt that these changes were due to severe anemia.

Our findings are contrary to those of Tsujimura,[6] who did not find any changes in the laryngeal muscles after passive hyperemia of the laryngeal veins. Of course, one must consider that the laryngeal muscles react differently to disturbances in the arterial influx of the blood than to passive venous hyperemia.

The last point to be discussed is the fact that in all of our cases the abductor muscle of the larynx was more affected than the adductor muscles. The number of our cases is too small to permit us to draw general conclusions; however, it is remarkable that in all of our cases this was observed. The observation is of practical importance, because we know from the famous law of Rosenbach and Semon that in recurrent nerve paralysis the abductors are always the first to be affected. Many years ago Grabower[7] found that the abductors of the larynx contained

6. Tsujimura, I. N.: Experimentelle Studien über die Veränderungen des Kehlkopfes durch venöse Stauung, Mitt. a. d. med. Akad. zu Kioto **18**:205, 1936.

7. Grabower, A.: Ueber die Degeneration funktionell gelähmter Kehlkopfmuskeln und die "Inaktivitätsatrophie," Arch. f. Laryng. u. Rhin. **28**:351, 1914; Die Verteilung und Zahl der Nervenfasern in den Kehlkopfmuskeln und die Hinfalligkeit des Erweiterers der Stimmritze, ibid. **16**:189, 1904.

fewer nerve fibers than the other muscles, a fact which should help explain the statements of Rosenbach and Semon. We observed the nerves within the muscles of our specimens and were able to confirm the findings of Grabower. In addition to this we noted that the abductor muscles seemed to be more sensitive to vasomotor disturbances than the other muscles; we consider this fact as a further argument to explain the law of Rosenbach and Semon.

NASOPHARYNGEAL FIBROMA

REPORT OF A CASE

THOMAS E. HUGHES, M.D.
RICHMOND, VA.

Many physicians with varied clinical experience have never encountered a fibroma of the nasopharynx. It is, therefore, a relatively rare tumor, yet a number of case reports are found in the literature.

Goldsmith [1] described the growth succinctly as a tumor formation histologically nonmalignant but pursuing clinically a malignant course, occurring almost entirely in young males and definitely retrogressing when the patient reaches the approximate age of 23, unique among tissue growths and found alone in the nasopharynx. Females are not, however, immune.

Ewing [2] said that the tumor usually appears as a firm, pale red mass attached by a broad base in the nasopharynx and not freely movable. The main mass of the tumor consists of dense fibrous and elastic tissue, containing numerous large, thin-walled blood vessels, located more abundantly near the free portion of the tumor. The cells are round, spindle-shaped or star-shaped fibroblasts, which are rather scanty except in certain foci of young connective tissue.

Pierce [3] stated that at times the vascularity is so rich that the tumor resembles cavernous tissue. It is covered with a pale, bluish red or red mucous membrane but has no definite capsule. Milligan,[4] Tilley [5] and Goldsmith [1] concurred with the statement of Pierce. The color and density vary with the vascularity. There are instances in which the tumor has been present for several years without producing serious trouble. It usually grows rapidly, however, and disturbs various functions by pressure rather than by invasion. Secondary anemia, ulcer-

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1. Goldsmith, P. G.: Nasopharyngeal Fibroma: A Clinical and Pathological Study, J. Laryng. & Otol. **38**:565-574 (Nov.) 1923.

2. Ewing, J.: Fibroma, in Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, pp. 180-182.

3. Pierce, N. H.: Typical Postnasal Fibromas and Their Spontaneous Disappearance, with Report of Case, Tr. Am. Laryng. A., 1911, pp. 119-127.

4. Milligan, W.: Nasopharyngeal Growths, J. Laryng. & Otol. **39**:537-544 (Oct.) 1924.

5. Tilley, H.: Diseases of the Nose and Throat, ed. 4, New York, Paul B. Hoeber, 1921, pp. 325-332.

ation and local infection are found as a result of frequent bleeding. These complications may prove fatal, or cerebral complications may cause death.

The average age of patients afflicted with nasopharyngeal fibroma is from 10 to 25 years. All of Pierce's [3] patients except 1, who was 31 years old, came within these limits. The age of Chamberlin's [6] patients varied from 10 to 28 years. In the series of 32 cases of New and Figi,[7] the ages of the patients were from 10 to 31 years, the average being 18½ years. Almost all observers have stated that there is a marked tendency of nasopharyngeal fibroma to atrophy when the patient reaches the approximate age of 25. Milligan [4] expressed the belief that this natural tendency toward regression is due to ossification of the sutural lines at the age of adolescence. Persky [8] and others claimed, however, that no satisfactory explanation has been given for this peculiar characteristic.

Scal [9] in 1927 reported the cure by radium of a nasopharyngeal fibroma in a woman 56 years old. Microscopically, the specimen showed almost pure fibrous tissue, rich in blood vessels.

A preponderance of the tumors reported as nasopharyngeal fibroma have occurred in males, though no apparent cause for such a predilection has been found. New and Figi [7] reported 28 males and only 4 females in their series of patients at the Mayo Clinic. Schleglmünig [10] reported the occurrence of nasopharyngeal fibroma in a girl and cited from the literature a few other occurrences of this growth in females. Bensch [11] stated that the development of the bony skull is arrested earlier in girls than in boys and that for this reason fibroma usually develops in boys; if it does appear in girls it becomes retrogressive at an early age.

Milligan [4] said that he had never seen multiple fibroma; Ballenger [12] claimed that fibroma is usually single, and Pierce [3] stated that it is

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6. Chamberlin, W. B.: Fibroma of the Nasopharynx, with Report of Four Cases, Ann. Otol., Rhin. & Laryng. **20**:683-693, 1911.

7. New, G. B., and Figi, F. A.: Treatment of Fibromas of the Nasopharynx: Report of Thirty-Two Cases, Ann. Otol., Rhin. & Laryng. **34**:191-196 (March) 1925.

8. Persky, A. H.: Fibroma Treated by Operation and Followed by Radium: Two Cases, Laryngoscope **39**:460-466 (July) 1929.

9. Scal, J. C.: Nasopharyngeal Tumor Cured with Radium: Case, M. J. & Rec. **125**:333-334 (March 2) 1927.

10. Schleglmünig, J.: Ueber ein typisches Nasenrachenfibrom bei einem Mädchen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **17**:1-6, 1926.

11. Bensch, H.: Die Nasenrachen-Tumoren Resp. Nasenrachen-Polypen, in Voltolini, R.: Die Krankheiten der Nase und des Nasenrachenraumes, Breslau, E. Morgenstern, 1888, pp. 371-401.

12. Ballenger, W. L.: Diseases of the Ear, Nose and Throat, ed. 2, Philadelphia, Lea & Febiger, 1909, pp. 356-359.

never multiple. Yet, in 1918 Coates [13] reported a case in which he found simultaneously a tumor in the nose and one in the nasopharynx, both diagnosed fibroma. Goldsmith found one large and two smaller tumors in 1 of his patients, all of which were fibroma. Nevertheless, instances of multiplicity are rare.

The exact point of origin of nasopharyngeal fibroma is often difficult to determine and is still a question of controversy. The opinion held by many observers is that fibroma may arise from the vomer, the inner surface of the internal pterygoid process, the front surface of the upper cervical vertebrae or, in fact, from any part of the roof or the lateral walls of the nasopharyngeal space.

Mackenzie [14] cited Nelaton as saying that the primary point of origin in all cases is the periosteum covering a limited area on the under surface of the base of the skull and corresponding to the basilar process of the occipital bone and the body of the sphenoid bone. He maintained that any other point to which the tumor appears to be attached, either in the nasopharynx or in the nose, is a point where secondary adhesions have formed from contact, either by pressure from or by ulceration of the tumor.

Chamberlin [6] asserted that the exact point and extent of attachment are difficult to determine because of the fact that only the under surface of the tumor can be seen with the mirror and that palpation, likewise, gives little information in this regard.

Jacques [15] stated that instead of the classic theory of a basilar origin and a primary pharyngeal seat for juvenile hemorrhagic fibroma there has been a tendency to substitute a new theory: namely, that the sphenoethmoid insertion is the primary seat of this neoplasm. In order to substantiate this point, he presented the fact that in the early stage of the disease only one choana is obstructed. The opposite side becomes obstructed subsequently if at all. In 1908 he presented before the French Laryngological Society the histories of 6 patients on whom he had operated and whom he had cured. In all of these the fibroma arose from the anterior wall of the body of the sphenoid.

Coates [13] claimed that it seems logical that tumors of the type under discussion have their origin in either the nose or the nasopharynx and that they are identical except in the location of their point of insertion.

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13. Coates, G. M.: Nasal and Nasopharyngeal Fibromata with Reference to Multiplicity of Growths, Ann. Otol., Rhin. & Laryng. **27**:191-205 (March) 1918.

14. Mackenzie, M.: Fibrous Polypi of the Nasopharynx, in A Manual of Diseases of the Throat and Nose, Philadelphia, P. Blakiston, Son & Co., 1884, vol. 2, p. 497.

15. Jacques, D.: Insertion and Seat of Rhinopharyngeal Fibromata, Ann. Otol., Rhin. & Laryng. **20**:881-892 (Dec.) 1911.

Casselberry [16] expressed the belief that growths in the nose and nasopharynx correspond in type to the structural elements of the tissue from which they originate and stated that the fibrous layer had been shown by Panas to be especially abundant and more densely fibrous at the upper and posterior part of the septum and at the adjoining space on the base of the skull. This accounts for the frequency of nasopharyngeal fibroma. In the anterior nares the fibrous tissue is less abundant and, therefore, fibroma is seldom found in this location.

No definite cause of fibroma has been found. In some instances a history of trauma has been reported, and in others infection has seemed to be a contributing factor. Pierce [3] said that there must be some etiologic factor, as proved by the relative infrequency of the growth, and that this factor may be found in the abnormal craniopharyngeal duct inclusion. Casselberry [16] stated the opinion that a local irritation from trauma may serve to excite hyperplasia of the fibrous elements and that in other instances a chronic inflammatory process may be the exciting cause. Ewing [2] has observed that chronic productive inflammation and fibroma are similar in histologic structure. Yet, characteristic examples of each are quite different.

The boy whose case I am reporting gave a history of concussion four years before the onset of symptoms. He also had sinusitis at the time the growth was discovered. By obstructing the nose, however, the tumor may have been a causative factor in the sinusitis rather than a result of it. Bensch [11] claimed that tumors of this type are due to the fact that the periosteum in a limited zone on the base of the skull is unable to produce bone tissue and that the surplus of nutritive substances is used in the development of a hypertrophic condition.

Nasopharyngeal fibroma is seldom discovered in its early stage, at which time it produces no symptoms. For this reason it would be discovered only on routine examination. A routine posterior rhinoscopic examination is, therefore, of great importance. With this procedure a growth may be located before serious symptoms develop. Nasal obstruction and nasal bleeding are probably the most frequent conditions that induce a patient with a nasopharyngeal growth to consult a physician. As the growth progresses other symptoms may develop: namely, secondary anemia from recurring hemorrhage, deafness, otitis media, obstruction in both nares, dyspnea, indistinct articulation, dysphagia, drowsiness and nasal discharge of a fetid character.

The subsequent symptoms depend on the route followed by the tumor. It may grow into one side of the nose and push the septum to the opposite side, or it may advance into both nares and separate the nasal bones and the eyes, producing exophthalmos or the deformity

16. Casselberry, W. E.: Nasal Fibromata, J. A. M. A. **10**:477-481 (April 21) 1888.

known as "frog face." Sometimes one or more of the paranasal sinuses are invaded. If the growth projects through the pterygomaxillary fissure, it may produce swelling in the cheek or in the temporal region.

Pierce [8] stated that the first symptom of an extrapharyngeal fibroma may be neuralgia of the second branch of the fifth nerve, from pressure on the gasserian ganglion.

The usual symptoms of intracranial pressure appear when the growth enters the cranial cavity, either by pressure erosion through the base of the skull or through one of the foramens. Death results more frequently, however, from severe recurring hemorrhage and its sequelae.

The duration of symptoms in the series of 32 cases of New and Figi [7] varied from two months to six years.

According to the majority of observers, the tumor receives its nourishment through its main point of attachment and not through its secondary attachments.

Nasopharyngeal fibroma may be confused with the pseudo or ordinary mucous polypus, with the fibrous polypus or with sarcoma. Chamberlin [6] pointed out that the mucous polypus differs in consistency as well as in histologic structure from nasopharyngeal fibroma. On the other hand, the fibrous polypus resembles fibroma both in consistency and in histologic structure.

The prognosis with the fibrous polypus, however, is much better than that with nasopharyngeal fibroma. Zarniko is Chamberlin's [6] authority for the following differential points in diagnosis: 1. The pseudo, as well as the fibrous, polypus is always pedunculated and the pedicle is usually small; the true fibroma is almost never pedunculated. 2. The true fibroma bleeds easily and spontaneously; the pseudo and the fibrous polypus do not. 3. The tendency to rapid growth and to erosion of neighboring structures is far greater with the true fibroma than with the mucous or fibrous polypus.

The differential diagnosis from sarcoma is more difficult. Both bleed readily, and both, sarcoma by direct invasion and fibroma by pressure, involve neighboring structures. Microscopic examination of excised portions of the tumor usually confirms the diagnosis. This method, however, is sometimes uncertain if the section is taken from a cellular portion of the fibroma. Fibroma does not metastasize; it does not involve the lymph nodes nor does it produce cachexia. Sarcoma involves the lymph nodes, produces cachexia and metastasizes. Hence, microscopic examination of a cervical lymph node frequently clears the diagnosis.

Getchell [17] expressed the opinion that nasopharyngeal fibroma should not be classified with nasofibroma, because nasopharyngeal fibroma

17. Getchell, A. C.: Nasopharyngeal Fibromata, Tr. Am. Laryng. A., 1909, pp. 50-58.

almost invariably occurs in boys near the age of puberty and has a marked tendency toward regression when the patient reaches the approximate age of 25. Nasal fibroma, on the contrary, is found in persons of either sex and more often in those past middle life.

Crowe and Baylor [18] stated that without the clinical findings and without the microscopic examination of a large section of the tumor it may be impossible to determine whether the growth is a round cell sarcoma, angioma, angiosarcoma or cellular fibroma. The uncertainty of microscopic diagnosis is increased by the inflammatory reaction that occurs with any tumor of the upper air passages.

In the earlier cases of New and Figi,[7] tissue was removed for microscopic examination. Fibroma, fibroangioma and fibromyxoma were found. The removal of tissue for examination was discontinued, however, because of the bleeding which accompanied it. More recently the diagnosis has been based on the history, the hardness of the tumor, the age of the patient and the clinical picture.

Milligan [4] expressed the belief that it is difficult to distinguish between sarcoma and cellular fibroma.

It is the consensus of those who have discussed this subject that the nasopharyngeal fibroma has a marked tendency toward regression when the patient reaches the age of adolescence. Several theories have been advanced to explain this unusual phenomenon, but none of them has been accepted as entirely satisfactory. Bensch [11] stated the belief that the growth tends to disappear at the age when the sphenoid and the basilar process of the occipital bone become united and when ossification of the cervical vertebrae has been completed. Goldsmith [1] claimed that the fibroma is of periosteal origin and tends to stop growing and undergo retrogressive changes after the patient reaches the age of 23 years. At this time active periosteal growth has terminated, and the skull has reached maturity.

On June 25, 1910 Pierce [3] examined a man, a laborer 31 years old, whose skull had been fractured about two months previously. He found a hard tumor in the nasopharynx, diagnosed fibroma. While waiting to have made a snare suitable for its removal, he examined the patient again on July 9 and discovered that the tumor had decreased in size. The only treatment had been the administration of potassium iodide. No operation had been done. The patient was kept under observation until only a slight nodular thickening remained at the site of origin of the tumor.

Bensch [11] cited Gosselin's classic case. The patient was a man 22 years old. In spite of long-continued treatment and repeated opera-

18. Crowe, S. J., and Baylor, J. W.: Benign and Malignant Growths of the Nasopharynx and Treatment with Radium, Arch. Surg. **6**:429-488 (March) 1923.

tions, he was in such poor physical condition that Gosselin advised against further operations. An incomplete hemiplegia and exophthalmos on the right side had already developed. This was thought to be due to entrance of the growth into the skull. As his case was considered hopeless, the patient was permitted to leave the hospital on March 26, 1870. No subsequent treatment except that necessary for transient hemorrhage was given. On Feb. 16, 1871 the patient returned to see Gosselin. The paresis and exophthalmos had disappeared, and the tumor was entirely absorbed.

The methods of treatment of fibroma of the nasopharynx have been numerous. The improvement in procedure which has come about through the years makes one of the most interesting chapters in the history and development of this particular phase of medicine.

In the earliest form of treatment, according to Mackenzie,[14] attempts were made to dilate the orifice of the nostril with serpentaria, gentian and laminaria to gain access to the tumor. These procedures, however, proved of little practical value. He further stated: "In the case of hard polypi, Hippocrates directed that the nostril should be slit open in order that the tumor might be thoroughly extirpated and the roots afterward destroyed with the hot iron."

Before the time of specialization in this field of surgery, tumors of the nasopharynx were operated on by general surgeons. The radical, or preliminary, operation was performed in order to gain access to the tumor. This was accomplished by resection of the superior maxillary bone and entrance into the nasopharynx through the maxillary sinus, by separation of the nasal bones and division of the nasal septum or by division of the soft palate and, at times, the chiseling away of the hard palate.

After the tumor was reached through the external route, it was removed by one of several methods, according to the fancy of the operator. Cutting forceps, scissors, the cold snare and heavy grasping forceps were some of the instruments used for this purpose. Some operators applied a hot iron or other cautery to the stump.

According to the testimony of numerous surgeons regarding their results with the radical operation for nasopharyngeal fibroma, this operation was considered formidable and dangerous and was accompanied by a high rate of mortality.

It is questionable whether such a procedure was justified, when the results are compared with those of the later operative procedures, which are accomplished through the natural passages. The earlier type of operation was accompanied by severe shock and hemorrhage, either during the operation or immediately afterward. It was not unusual for the patient to die on the table. In many instances the external carotid artery was tied, and a tracheotomy was performed before the radical operation was begun.

As recently as 1921 Wall [19] operated on a patient with nasopharyngeal fibroma by Kocker's resection method. The results are not given.

In 1883 Lincoln [20] reviewed the results of treatment with the older operative methods in cases of nasopharyngeal fibroma and compiled a table of data on 74 operations by different surgeons. In 28 cases the bones of the face were resected to gain access to the nasopharynx for removal of the growth. In several instances the fibroma returned. This necessitated a repetition of the operation. In 8 cases, or 28 per cent, death followed operation immediately or in a few days.

He concluded that there were many cases in which nasopharyngeal fibroma could be treated successfully by a method simpler and safer than that usually employed by eminent surgeons. Lincoln [20] reported cases in which the fibroma was successfully treated by means of the galvanocautery écraseur, with subsequent destruction of the stump by means of the galvanocautery. This procedure was accomplished through a natural opening, either mouth or nose. It was also recommended by Ingals [21] and Delavan.[22] In the years 1891 to 1901 Delavan [22] reviewed the histories of 120 patients that had been operated on by the older methods and found a mortality rate of 25.9 per cent, with recurrences in 15.4 per cent. Among 66 patients that were treated with the more modern methods, there was not a fatality.

Other methods of treatment through the nose and mouth were employed. These were electrolysis, avulsion by means of a heavy cold wire snare or heavy forceps, excision, crushing, gouging, and removal by actual cautery or chemicals.

In 1905 Harmon Smith [23] completely destroyed a fibroma of the nasopharynx by injecting monochloroacetic acid into it. Lactic acid was used by other clinicians with success.

Five patients were operated on by New and Figi [7] early in their series by the avulsion method through the natural passages. Two deaths followed.

The avulsion method has been used by many operators, notably Coates [13] and Spielberg.[24] Either a heavy curved forceps was used or

19. Wall, G. A.: Juvenile Nasopharyngeal Fibroma: Report of Case Treated by Kocher's Osteo-Plastic Method, Laryngoscope **31**:287 (May) 1921.

20. Lincoln, R. P.: On Results of Treatment of Nasopharyngeal Fibromata, with Demonstration of Successful Cases, Together with Table of Seventy-Four Operations by Different Surgeons, Arch. Laryng. **4**:258-281, 1883.

21. Ingals, E. F.: Removal of Nasopharyngeal Fibromata by the Galvano-Cautery or Cold Wire Écraseur, J. A. M. A. **2**:533-535 (May 17) 1884.

22. Delavan, D. B.: Nasopharyngeal Fibroma, Tr. Am. Laryng. A., 1911, pp. 15-28.

23. Smith, H., in discussion on Delavan.[22]

24. Spielberg, W.: Fibroma: Two Cases, Laryngoscope **42**:872-876 (Nov.) 1932.

heavy piano wire, with which a loop was made to encircle the growth and to pull it loose from its attachment. These instruments may be used alone or in conjunction with diathermy or radium.

The use of radium and of roentgen rays in the treatment of fibroma of the nasopharynx is the most recent advance in the therapy of this disease.

In 1921 Dabney [25] treated with radium a nasopharyngeal fibroma in a boy 15 years old. The patient was in a critical condition from loss of blood. He showed miraculous improvement after the first treatment and rapidly regained his health.

Moffat and Jones-Phillipson [26] in 1924 had a patient in whom proptosis had developed as a result of extension of a fibroma of the nasopharynx. The condition was successfully treated with radium and surgical diathermy.

New and Figi [7] described three methods of radium treatment: 1. A large tube or capsule containing 50 mg. of radium was applied directly in the nares for ten to fifteen hours. 2. Steel points containing the radium emanations were used. 3. The element was inserted into the tumor. The treatment was repeated in six weeks to two or three months, depending on the reaction of the patient and the result of the previous treatment. The number of treatments in each case varied from two to nine. The dosage of the secondary treatments varied according to the progress of the patient. In most of their patients the tumor cleared up entirely; in the others it was held in check during its period of activity.

Crowe and Baylor [18] reported, in 1923, 4 cases in which nasopharyngeal fibroma was treated with radium and by partial surgical removal. The emanations were implanted directly into the tumor, and irradiation was also given intranasally and externally. Two of their patients were well and the other 2 had improved at the time of their report.

In a patient with recurring severe hemorrhage complicating fibroma of the nasopharynx, Clark [27] ligated and severed the external carotid artery and the external and the internal jugular vein. Then four radium and eight roentgen treatments were given over a period of six months. This procedure was followed by complete disappearance of the tumor.

Keith and Keith [28] inserted four radium needles, 12.5 mg. each, directly into a nasopharyngeal fibroma, about 1 cm. apart, and allowed

25. Dabney, S. G.: Fibroma of the Nasopharynx, Kentucky M. J. **21**:228-229 (May) 1923.

26. Moffat, H. A., and Jones-Phillipson, C. E.: Nasopharyngeal Fibroma Treated with Radium and Surgical Diathermy, J. Laryng. & Otol. **39**:571-573 (Oct.) 1924.

27. Clark, A. F.: Nasopharyngeal Fibroma, Texas State J. Med. **21**:658-660 (March) 1926.

28. Keith, D. Y., and Keith, J. P.: Juvenile Nasopharyngeal Fibroma Treated with Radium with Report of Case, Kentucky M. J. **23**:26-29 (Jan.) 1925.

these needles to remain for about twelve hours. In addition, a radium capsule was inserted into the nasopharynx, and extensive high voltage roentgen therapy was employed. In two months the growth had entirely disappeared.

Others who have used radium with favorable results, either alone or in conjunction with operative methods, are Milligan,[4] Persky,[8] Mertins,[29] Pryor [30] and Forster.[31]

Milligan [4] employed the emanations, using twelve to eighteen tubes inserted into the mass. Mertins [29] said he considered 50 milligram hours of radium the average dose to be used for each cubic centimeter of tumor tissue. Pryor [30] used radium needles in the treatment of a fibroma of the nasopharynx. The dosage was repeated every two or three months for a year. A total of 1,270 milligram hours was given. The mass had entirely disappeared in one and one-half years after the first treatment. Forster's [31] patient was treated successfully with radium, diathermic coagulation and roentgen rays.

REPORT OF A CASE

L. A., a 19 year old youth, was first examined by me in December 1937. His complaint was complete obstruction of the right nostril and occasional nosebleed. The family history was noncontributory. His tonsils and adenoids had been removed at the age of 5. In 1931 he fell from a tree and sustained a concussion. In 1935 he received a blow on the head while playing football and was unconscious for fifteen hours. About a year later headache developed, which was relieved after several local treatments to his nose. At that time he had slight difficulty in breathing through the right naris, but the cause of the obstruction was not discovered.

While breathing through the left naris was normal, slight difficulty in breathing through the right naris had been noticeable for about two years. The obstruction gradually increased until this side of the nose became completely blocked and filled with secretion. Bleeding from the right naris had occurred at intervals for about a year. This was spontaneous and sometimes began during sleep. At times the right ear felt stopped.

The patient talked as though his nose were obstructed. When he swallowed, the obstruction was more noticeable. He was a well built, naturally strong young man, apparently in good health except for the complaint from which he sought relief. There was some evidence of anemia due to frequent epistaxis. The lymph nodes were not palpable. There was no facial enlargement and no abnormality in expression.

A mucopurulent discharge was seen in the right naris, and the mucous membrane was edematous. The choana of this side was blocked by a mass, the surface

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29. Mertins, R. S.: Radium Treatment of Fibromata, South. M. J. **21**:711-713 (Sept.) 1928.

30. Pryor, W. R.: Fibroma: Two Cases, Kentucky M. J. **33**:387-390 (Aug.) 1935.

31. Forster, H. V.: Advanced Case of Fibroma Treated by Diathermy Coagulation and X-Rays, J. Laryng. & Otol. **46**:402-404 (June) 1931.

of which seemed smooth, reddish and resistant. With the nasopharyngeal mirror, the mass was easily seen. It almost filled the right nasopharynx and extended slightly into the left side, though not sufficiently to obstruct breathing on this side. Palpation through the mouth revealed that the tumor was smooth, firm and only slightly movable. Its attachment could not be definitely located. It was, however, broad and not pedunculated.

Transillumination and roentgen examination showed the right maxillary sinus to be cloudy. Pus was washed out through the natural ostium, and the infection subsided after a few irrigations.

With the patient under local anesthesia, a piece of the growth was removed through the nose for biopsy. Slight bleeding followed. Histologic examination

Nasopharyngeal fibroma (L. A., aged 19).

of the specimen showed it to be a fibroma, heavily infected. On Dec. 21, with the patient under anesthesia induced by ether, the first operation was performed. Two rubber catheters were passed through the nose and brought out through the mouth for retraction of the soft palate and for use in the insertion of a postnasal plug. Traction was made on the soft palate sufficiently to expose to view the lower end of the growth. An Eve tonsil snare with heavy wire was used to engage the growth, in an effort to pull it away from its attachment. A mass about ¾ inch (2 cm.) in diameter was removed, which was thought to be the entire tumor. As the mass could not be cut through by means of the wire, it was pulled loose with force. Profuse bleeding occurred, which required the immediate use of a postnasal plug. As there was no more bleeding, the postnasal plug was removed the following day, and the patient left the hospital. On subsequent examination

with the postnasal mirror, it was discovered that the larger portion of the tumor still remained and that only a smaller lobe had been detached.

In a few days the boy was allowed to return to school in another city. The operation was completed on March 25, 1938, with the patient under general anesthesia.

A cold wire snare was devised with the use of heavy piano wire and the lumen of an Eve snare with stilet removed. Both ends of a long piece of wire were passed through the lumen of the instrument until only a small loop remained at the distal end. The instrument was passed into the nose until the tumor was reached. The wire loop was then fed into the nasopharynx until it could be seen under the retracted soft palate. The ends of the wire were gradually pulled up while the wire loop was directed around the tumor with the forefinger, which was inserted through the mouth. The wire was made taut by wrapping the ends around an artery clamp, which was used as a handle for traction. The tumor was pulled away from its attachment and removed through the nose, still engaged in the wire snare. The wire had grasped the tumor about its center and did not slip. Neither did it cut into the growth, notwithstanding the fact that much force was used in detaching it. The growth was 1½ inches (3.8 cm.) in diameter, smooth, firm and bluish red. The two pieces removed are believed to have come from the same tumor. There was profuse bleeding as soon as the tumor was removed, which was readily controlled with a postnasal plug held by heavy silk, extending through the nose and mouth. Unfortunately, during the night the plug became slightly loosened. This was followed by severe bleeding. Although the patient's condition was not critical, a blood transfusion was given and physiologic solution of sodium chloride was administered.

The postnasal plug was removed in about forty-eight hours, and the patient left the hospital at the end of a week and returned to school several days later.

He was examined in June 1938 and again in September 1939, one and one-half years after the operation. During this time there had been no nasal obstruction or bleeding. No tumor was visible. Before the operation the definite location of the attachment could not be made out. At the last examination, however, the postnasal mirror showed an area of scar tissue, about ½ inch (1.3 cm.) in diameter, on the basilar process of the occipital bone and on the base of the vomer, more on the right side than on the left. Undoubtedly this area of scar tissue was the site of the attachment of the fibroma.

SUMMARY

A review of the literature on fibroma of the nasopharynx shows that nothing new has been reported by recent writers except in the methods of treatment. Radium therapy seems to be the method of choice. It has proved superior to any other form of medical or surgical treatment.

THE ACUTELY INVOLVED MASTOID (WITHOUT COMPLICATIONS) BEFORE AND AFTER OPERATION

A CLINICAL AND ROENTGENOLOGIC STUDY

CHARLES W. BARKHORN, M.D.
NEWARK, N. J.

There has been considerable study of acute, uncomplicated involvement of the mastoid in its preoperative and operative stages,[1] and a tremendous amount of literature has been published on the subject. The clinical signs and symptoms of mastoiditis, a study of roentgenograms and a combined discussion by both the roentgenologist and the otologist [2] have been a topic of interest. Law [3] has written much in both periodical and book form on the roentgenologic aspects of mastoids. Granger's [4] book on sinuses and mastoids is popular, and numerous other otologists [5] have devoted much time and labor to the subject; however, the study stopped there. The literature contains little on the roentgenologic aspect of the mastoid which has been operated on for acute uncomplicated disease. When complications such as sinus thrombosis and brain abscess occur, additional roentgenograms are required, but as to what the normal postoperative roentgenogram of the mastoid discloses there is little to be found.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., June 1940.

1. Kreutz, G. C., and Whitter, G. L.: Observations in Three Hundred Cases of Acute Mastoiditis, Ann. Otol., Rhin. & Laryng. **46**:1060-1064 (Dec.) 1937.

2. Berg, H. M., and Constans, G. M.: The Clinical, Roentgenological and Operative Findings in One Hundred and Fifty-Eight Cases of Mastoiditis, Am. J. Roentgenol. **30**:452-457 (Oct.) 1933.

3. Law, F. M.: (*a*) Roentgen Examination of the Mastoid Process, Am. J. Roentgenol. **31**:482-486 (April) 1934; (*b*) Mastoids, Roentgenologically Considered, ed. 2, New York, Paul B. Hoeber, 1929.

4. Granger, A.: Radiological Study of the Para-Nasal Sinuses and Mastoids, Philadelphia, Lea & Febiger, 1932.

5. (*a*) Adams, W. S.; Cawthorne, T., and Mitman, L.: Discussion on the Value of Radiology in Diseases of the Ear, Proc. Roy. Soc. Med. **29**:969-980 (June) 1936. (*b*) Schillinger, R.: Roentgenologic Aspects of Mastoiditis, Am. J. Roentgenol. **39**:193-201 (Feb.) 1938. (*c*) Schumacher, F. L.: The Roentgenologic Study of the Mastoid Area, Radiology **27**:717-721 (Dec.) 1936.

H. K. Taylor,[6b] of the roentgen ray department of the Beth Israel Hospital, in New York, in a discussion of roentgenograms of mastoids stated:

> . . . in an operative mastoid no cellular structures are demonstrated. The operative field is clearly visible, demarcated by the sharp linear shadows of the tegmen and the sinus. The necessity for roentgenographing post-operative cases arises only when re-infection of the mastoid occurs and when a wound presents a persistent discharge. If any cells were not removed at the time of operation, they will be demonstrated on a flat plate. For a more detailed examination, stereoscopic plates should be taken, particularly when a sequestrum is present.

This statement, so far as I am able to find in the literature, is practically all that has been written concerning postoperative roentgenograms of the mastoid. There has been no published description or discussion of the roentgenologic picture of the affected area after successful mastoidectomy for one to compare with that obtained after the unsuccessful operation. Therefore, in this paper I will attempt to discuss both the preoperative and the postoperative roentgenologic aspects of the mastoid bone. I will include in the discussion the clinical onset of the middle ear disease and the preoperative roentgenograms and will correlate the findings with the observations at operation and summarize the postoperative clinical course; finally, I will describe particularly the postoperative roentgenograms of the satisfactorily healed mastoid taken some years after surgical intervention. I will present this picture for comparison with that obtained when the results are unsatisfactory.

Almour,[7] in a paper on the nonhealing of mastoid wounds, stated:

> The local factors, exclusive of inadequate surgery, in the nonhealing of mastoid wounds comprise, (1) excessive widening of the tympanic antrum, (2) excessive smoothening of the operative cavity, (3) improper management of the zygomatic root, (4) continuing necrosis of the inner table and (5) suppurations beyond the confines of the mastoid process.

My feeling is that if these points can be illustrated in postoperative roentgenograms of mastoids and shown to have been dealt with properly, then mastoid bones in cases in which operative results were unsatisfactory can be reexamined and the areas of trouble more easily recognized.

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6. (*a*) Deutsch, L., and Mayer, E. G.: The Roentgen Examination of Resected Mastoids, Fortschr. a. d. Geb. d. Röntgenstrahlen **33**:887-889, 1925; abstracted, Am. J. Roentgenol. **16**:279 (Sept.) 1926. (*b*) Taylor, H. K.: The Roentgenogram in Mastoid Diseases, ibid. **19**:522-530 (June) 1928.

7. Almour, R.: Non-Healing of Mastoid Wounds, New York State J. Med. **36**:1097-1100 (Aug.) 1936.

MATERIAL AND METHOD

To advance this study I have worked in cooperation with two roentgenologists, Drs. Charles F. Baker and W. James Marquis, and as subjects for study we chose 24 patients on whom I had performed mastoid operations. We selected those in whose cases as complete records as possible had been kept from the onset of infection to operation and then postoperatively until we reexamined the patients roentgenologically. The original roentgen films were reviewed by the roentgenologists and by me; the operations were all performed and the patients cared for postoperatively by me; finally, the postoperative roentgenograms were examined and discussed by both roentgenologists and by me just before the writing of this paper.

Of the 24 patients chosen for study, there were 4 under 5 years of age, 9 between the ages of 5 and 10 years, 2 between 10 and 20, 3 between 20 and 30, 1 between 30 and 40, 4 between 40 and 50 and 1 between 50 and 60 years of age. There were 12 females and 12 males. Twenty-one patients had unilateral mastoiditis, 9 on the right and 12 on the left side, and 3 had bilateral mastoiditis. All aural symptoms began as the result of grip, acute coryza or sore throat, and from one to fourteen days elapsed after the onset of the predisposing conditions before either the ear drum ruptured spontaneously or a paracentesis was required. In 10 of the cases the ear ruptured spontaneously, in 13 a paracentesis was necessary and in 1 case the right ear ruptured spontaneously and the left required opening. All patients presented the conventional signs of mastoiditis, such as continuous profuse purulent aural discharge without signs of lessening, continued pain and headache, particularly at night, continued elevation or fluctuation of the temperature, postauricular tenderness or edema, narrowing or funneling of the external auditory canal and diminution of hearing. In addition to this, there was a positive indication for operation in all cases, as shown by the roentgenograms.[8]

Only 1 patient was operated on during the first week of symptoms; 8 were operated on after two weeks of continued and increasing signs, and the remaining 15 were not operated on until symptoms had been present three or more weeks. The longest interval between onset of aural trouble and operation was nine weeks. It has been my contention that only in rare cases should a mastoid operation be undertaken during the first week of symptoms. Unless untoward complications appear, I feel it better to wait two or even, by choice, three weeks; then if the ear is still discharging and not showing signs of improving, mastoidectomy must be undertaken.

OBSERVATIONS

In the study of the roentgenograms made prior to operation, the following points were noted:

1. The type of mastoid, whether infantile, pneumatic or sclerotic.

2. The size of the mastoid, whether it was large or small, whether the cells extended far into the zygomatic region or whether there were postsinal cells.

3. The middle ear canal, whether it appeared clear or occluded. This, in our opinion, indicated proper roentgen technic for superimposing external and internal auditory meatuses.

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8. Brownell, D. H., and Hauser, I. J.: The Roentgenogram as an Aid in the Diagnosis of Surgical Mastoiditis, Ann. Otol., Rhin. & Laryng. **47**:240-246 (March) 1938.

4. The antral area in the lateral view, whether it appeared large, small, dense or cloudy. This indication we found to be of considerable importance in predicting possible erosion of the sinal plate.

5. The presence of distinct cell layers,[9] whether the squamous and basal petrous portions of the temporal bone could be distinguished as two separate layers, disclosing involvement of either or both, or whether the cells were merely in a combination of large or small groups.

6. The trabeculae of the cells, whether they appeared thick, thin or hazy or were absent. In conjunction with the trabeculae we noted whether the cells themselves were blurred, dense or coalesced.

7. The visibility of the sinal plate, whether there was any break in continuity, indicating possible erosion of bone.

8. Finally, any change in the outline or density of the petrous apex was observed.

With this outline in mind, we examined the preoperative roentgenograms of the selected patients and found that even though the ages of the patients extended from 2 to 58 years, only two infantile[10] mastoids were identified by the roentgenograms. None of the mastoids was sclerotic.[9a] Twenty-two of the remaining ones were of the pneumatic[9a] type, made up of large or small cells or of a combination of cells of both sizes. Ten mastoids were large, with cells extending into the zygomatic region. Fourteen of the mastoids were of the small type, without zygomatic or postsinal cellular involvement. The middle ear canal appeared to be clear in the roentgenograms of 13 patients and tended to be occluded in those of 11. This fact we considered to be due to a technical error, and it gave us no hint as to pathologic conditions within the ear. An increase in density in the antral region was of distinct importance in our opinion. This region was best seen in the film made in the lateral position. In 11 of our cases there was indication of increased density. In the remaining 13 cases the density was not increased. In further considering the cases in which there was an increase of density, we found that in 3 a perisinal abscess was present, in 2 of which the abscess was identified before operation, whereas among the 13 remaining cases, in which the antrum was not dense, there were 2 in which a perisinal abscess was present, but in only 1 was its presence suspected before operation. It would seem from these observations that in cases in which the antral region appeared dense the presence of a perisinal abscess might be expected. The differentiation of cell layers[9] into squamous and

9. (a) Gerber, I.: Some Observations of Mastoid Structure as Revealed by Roentgen-Ray Examination, Am. J. Roentgenol. **6**:1-11 (Jan.) 1919. (*b*) Schumacher.[5c]

10. Crain, C. F.: Roentgenologic Considerations in Infant Mastoids, Am. J. Roentgenol. **38**:592-601 (Oct.) 1937. Gerber.[9a]

basal petrous portions as such was more difficult. These distinct layers, frequently seen at operation, could not be identified with certainty in all the roentgenograms. Our examination indicated only 4 cases in which we could be certain of a division. In the remaining cases the cells appeared as a combination of large or small cells. The trabeculae were found to be thickened in 15 of the cases, in 5 they were thin and in the remaining 4 no trabeculae were in evidence. The cells themselves were all blurred, indicating a stage of coalescence. The petrous apexes were found to be normal in all cases.

Operation disclosed coalescence of the mastoid in 20 patients. In 3 of them coalescence of the mastoid was combined with marked sclerosis in the region of the antrum, and 2 of these patients had perisinal abscesses. There was one hemorrhagic mastoid. A total of 5 patients showed erosion of the sinus plate, 2 of whom had extensive granulations on the sinal wall, extending beneath the angle of the superior petrosal sinus (sinodural angle) and involving the dura. These patients required removal of this bony angle so as to insure adequate exposure beyond the granulations. As was previously stated, an appearance in the roentgenogram of increased density about the antral region led us to suspect the presence of perisinal abscess, and an abscess was disclosed at operation in 3 of these 5 patients.

All operations were performed according to an anatomic rather than an exclusively pathologic plan:

> The zygomatic region was thoroughly explored and the mastoid tip either removed or taken down to the level of the horizontal semicircular canal. All postsinal cells were thoroughly uncapped, and the facial ridge was narrowed to the limit of disease, but the ridge was not lowered for fear of causing collapse of the external canal. The closure of the mastoidectomy wound was routine, a 2 inch (5 cm.) Penrose drain being inserted into the antrum and brought out the lower end of the wound. In addition to this, about 14 inches (35.5 cm.) of ½ inch (1.27 cm.) selvaged petrolatum gauze was folded accordion-like into the mastoidectomy cavity, and its folds were left protruding at the lower end of the incision. The purpose of this drain was to prevent the formation of a septic blood clot. Two loosely placed no. 1 plain catgut sutures approximated the periosteum and the muscle, and the skin was closed with two black silk sutures, or skin clips, and an alcohol dressing was applied. At the end of three days all wounds were dressed, one-half the petrolatum gauze drain being removed and a clean dry external dressing applied. On the fifth day, the remaining gauze drain and the clips, or sutures, were removed, but the Penrose tubing was left in place. The wounds were dressed every other day, and the Penrose tubing was removed when the middle ear became dry, or at about the end of two weeks. After removal of the drain, the external dressing was removed and the patient instructed to protect the mastoidectomy incision and the middle ear with cotton covered with 1 per cent yellow mercuric oxide ointment, which was to be changed twice a day.

A report on the cultures of material from the mastoids showed the hemolytic streptococcus to be the most common offender. It was

present in 15 of the cases, whereas a nonhemolytic streptococcus was present in only 3 cases. There were 4 cases in which culture of the material showed undifferentiated pneumococci and 1 in which it yielded Streptococcus viridans, and the report of one culture was, "no growth."

The average length of time for the patients to be hospitalized was seven days; they were discharged after the temperature remained normal for forty-eight or more hours. In all of the cases the hospital course was uneventful, with 1 exception, and in that case a local cutaneous irritation necessitated longer hospitalization. Postoperative recovery was uneventful except in 2 cases; in 1 of these pyelitis developed, a complication which lasted five weeks, and in the second case a reopening of the posterior incision was required, to eliminate a pocketing of purulent material. The average time required for the middle ear to dry and the drum to assume a normal appearance was two and one-half weeks, and the average time for complete healing was between five and six weeks. The hearing became normal in all but 2 cases, and in both of these the patients had had a previous aural condition which had affected the hearing before the reported condition; these 2 patients retained 50 per cent normal hearing.

In examining the healed incision, I noted a marked depression in only 1 case. The deep hole so common after early mastoidectomies was not present. There were 13 patients with a practically flat, or hair line, scar, with little postauricular depression. In 10 cases the depression was considered moderate. The aural canal showed evidence of collapse in 5 cases, slight in 4 and marked in 1, while in the other 19 cases the appearance of the external auditory meatus was normal. The ear drums were normal and intact in all but 1 case; in this instance there was a dry perforation as the result of previous otitis media.

Postoperative roentgenograms were made of the mastoid bones of all the 24 patients, and were studied in the hope of establishing normal criteria as a basis for comparison with roentgen findings in cases in which the proper tendency toward healing was not shown. The usual anterior-posterior and lateral exposures were made, and the most striking condition shown was the anatomic defect. In the anterior-posterior roentgenogram an acute, right or obtuse triangular area was in evidence, the superior border being made up of an extension of the mastoid tegmen laterally to the margin of the skull and the medial side being formed by a line drawn from the tegmen and passing through the fallopian canal of the facial nerve to the tip of the mastoid process. A line connecting the extremities of these two from the margin of the skull to the tip of the mastoid constituted the base. Within the bounds of this triangle, the operative defect of the mastoid bone was contained.

On examination of the lateral plate, another triangular defect was noted. This time the boundaries were: superiorly, the tegmen of the antrum, extending from the middle ear to the junction of the sinus and the dural plate; medially, a line from the tegmen, passing through the facial canal to the mastoid tip, and laterally, the curve of the sinus itself. This area, likewise, was a triangle, but with a base whose curve corresponded with that of the sinus.

Examination of the anterior-posterior triangular area furnished a view of the antral region as it encroached on the region of the superior semicircular canal, and in nearly all of the roentgenograms there was evidence of some remaining cells, either deep antral or perilabyrinthian. These cells, however, were all clear, with normal looking trabeculae, and showed no evidence of disease. They were the cells uncapped during the mastoid operation in the region of the lateral semicircular canal and the aditus and were purposely not too extensively removed, my feeling being that extensive curetting in this region was prone to produce too great a widening of the mastoid antrum, preventing adhesions and permitting reinfection and subsequent nonhealing of the mastoid. This opinion has been brought out by Almour [7] in his paper on nonhealing of mastoid wounds. There was no evidence of any remaining cells in the subantral or the tip region; a sharp acute angle, formed by the medial and lateral sides of the triangular defect, was evident in the roentgenogram; there was no evidence of the presence of cells at the superior lateral angle of the tegmen of the mastoid. In fact, except for the cells in the periantral region, this triangular defect appeared dark and free from densities that might obstruct the roentgen ray. Examination of the triangular defect portrayed by the lateral roentgenogram revealed more detail of the remaining cells in the antral region but showed them to be with clear outline and trabeculae. There was no evidence of cells in the subantral region or in the mastoid tip. The zygomatic region, which Almour [7] stressed as a source of nonhealing of the mastoid cavity, was free of all cells. The sinus plate, sharply outlined and extending from the sinodural angle to the mastoid tip, was clear and distinct. There was no evidence of any overlying or surrounding cells, and there was no evidence of any break in contour which might indicate necrosis of the plate except in the 2 cases in which the angle of the superior petrosal sinus had been removed at the operation. In these cases an entire absence of the plate was noted, but a difference in the density of shading outlined the contour of the sinus. In addition, the lateral exposure gave a picture of the area beyond the confines of the mastoid bone and displayed no evidence of any cells that might have been left at operation. This point is important in the nonhealing of mastoid wounds.

TABLE 1.—*Clinical History Before and After Operation*

Case; Patient	Age, Yr.; Sex	Onset	Ear Drum Ruptured Spontaneously or by Paracentesis	Duration Before Operation	Condition of Mastoid; Operation	Culture	Hospital Course	Postoperative Hospital Course	Time Required for Middle Ear to Become Dry	Time Required for Hearing Posteriorly	Hearing	Comment on Mastoidectomy Incision
1 A. J.	9 ♂	Cold and earache 1 wk.	Paracentesis, left	3 wk.	Coalescing; extensive simple mastoidectomy; perisinal abscess	Hemolytic streptococcus	Uneventful; temperature normal 5th day	Uneventful	2½ wk.	5 wk.	Normal	Moderate depression; no prolapse; drum intact
2 S. P.	5 ♂	Cold and earache 24 hr.	Paracentesis, right	2 wk.	Coalescing; extensive simple mastoidectomy	Hemolytic streptococcus	Uneventful; temperature normal 4th day	Uneventful	3½ wk.	4 wk.	Normal	Repeated otitis; no prolapse; slight depression; drum intact
3 S. P.	2 ♀	Grip 6 wk.; earache 2 wk.	Paracentesis, right	2 wk.	Coalescing; simple mastoidectomy	Hemolytic streptococcus	Stormy; temperature normal 11th day	Pyelitis, 5 wk.	2 wk.	5 wk.	Normal	No prolapse; slight depression; drum intact
4 S. V.	3 ♀	Cold and earache 4 wk.	Spontaneous, right	4 wk.	Coalescing; simple mastoidectomy	No growth	Uneventful; temperature normal 4th day	Uneventful	3 wk.	4 wk.	Normal	Slight depression; no prolapse; drum intact
5 N. G.	40 ♂	Cold 3 wk.; pain 2 wk., left ear	Spontaneous, left, 2 wk. before mastoidectomy	4 wk.	Coalescing; extensive simple mastoidectomy	Nonhemolytic streptococcus	Uneventful; temperature normal 3d day	Uneventful	3 wk.	4 wk.	50%; previous aural lesion	Dry perforation; moderate depression; no prolapse; drum intact
6 G. A.	4 ♂	Cold 6 wk.; pulsation	Spontaneous, left, 6 wk. before mastoidectomy	6 wk.	Sclerotic; extensive simple mastoidectomy; perisinal abscess	Str. viridans	Uneventful; temperature normal 6th day	Stormy; wound reopened	3 wk.	7 wk.	Normal	Ear noise; moderate depression; throbbing; no prolapse; drum intact
7 C. A.	16 ♀	Grip 5 wk.; earache, right	Spontaneous, right, 5 wk. before mastoidectomy	5 wk.	Coalescing; extensive simple mastoidectomy; perisinal abscess	Hemolytic streptococcus	Uneventful; temperature normal 3d day	Uneventful	2 wk.	3 wk.	Normal	Slight depression; no prolapse; drum intact
8 H. M.	44 ♀	Grip and pain in left ear 3 days	Paracentesis, left	4 wk.	Coalescing; extensive simple mastoidectomy	Nonhemolytic streptococcus	Uneventful; temperature normal 6th day	Uneventful	2 wk.	5 wk.	Poor, 50%	Moderate depression, left; no prolapse; drum intact

H. M.	♀	Grip and pain in left ear 3 days	Paracentesis, left	4 wk.	Coalescing; extensive simple mastoidectomy	Nonhemolytic streptococcus	Uneventful; temperature normal 6th day	Uneventful	2 wk.	5 wk.	Poor, 50%	Moderate depression, left; no prolapse; drum intact

9 McG. E.	21 ♀	Cold 7 wk.; no earache	Spontaneous, right, 7 wk. before mastoidectomy	7 wk.	Coalescing; extensive simple mastoidectomy; perisinal abscess	Hemolytic streptococcus	Uneventful; temperature normal 3d day	Uneventful	2½ wk.	5 wk.	Normal	No depression; no prolapse; drum intact
10 W. N.	6 ♀	Cold and earache 3 wk.	Spontaneous, right and left	3 wk.	Coalescing; double mastoidectomy	Hemolytic streptococcus	Uneventful; temperature normal 3d day	Uneventful; labyrinthitis 1 mo. later	2½ wk.	4 wk.	Normal	Slight depression; no prolapse; drum intact
11 R. J.	5 ♂	Cold 7 wk.; repeated abscess	Paracentesis, left, 7 wk.; reopened 1 wk. before mastoidectomy	7 wk.	Coalescing; extensive simple mastoidectomy; perisinal abscess; angle removed	Pneumococcus	Uneventful; temperature normal 4th day	Uneventful	3 wk.	6 wk.	Normal	Slight depression; no prolapse; drum intact
12 S. A.	29 ♂	Cold 5 wk.; earache 3 wk.	Paracentesis, right	3 wk.	Coalescing; simple mastoidectomy	Pneumococcus	Uneventful; temperature normal 3d day	Uneventful	2½ wk.	4 wk.	Normal	Slight depression; no prolapse; drum intact
13 M. A.	5 ♀	Cold 1 wk.; aural pain 3 days	Spontaneous, left	5 days	Hemorrhagic; simple mastoidectomy	Hemolytic streptococcus	Uneventful; temperature normal 6th day	Uneventful	2½ wk.	5 wk.	Normal	Slight depression; slight prolapse; drum intact
14 K. E.	38 ♂	Cold 2 wk.; earache 2 wk.	Spontaneous, right	2 wk.	Coalescing; extensive simple mastoidectomy	Pneumococcus	Uneventful; temperature normal 4th day	Uneventful	4 wk.	7 wk.	Normal	Slight depression; slight prolapse; drum intact
15 S. J.	6 ♀	Cold and earache 2½ wk.	Spontaneous, right	4½ wk.	Coalescing; extensive simple mastoidectomy	Hemolytic streptococcus	Stormy; preoperative, 2 wk.; uneventful postoperative; temperature normal 7th day	Uneventful	5 wk.	6 wk.	Normal	Moderate depression; no prolapse; drum intact
16 S. R.	5 ♂	Cold 2 wk.; earache	Paracentesis, left, 10 days before mastoidectomy	2 wk.	Coalescing; simple mastoidectomy	Hemolytic streptococcus	Moderately stormy preoperative; uneventful postoperative; temperature normal 3d day	Slow	4 wk.	9 wk.	Normal	Bony chips extruded through scar after 6 wk.; moderate depression; slight prolapse; drum intact

TABLE 1.—*Clinical History Before and After Operation—Continued*

Case; Patient	Age, Yr.; Sex	Onset	Ear Drum Ruptured Spontaneously or by Paracentesis	Duration Before Operation	Condition of Mastoid; Operation	Culture	Hospital Course	Postoperative Hospital Course	Time Required for Middle Ear to Become Dry	Time Required for Hearing Posteriorly	Hearing	Comment on Mastoidectomy Incision
17 M. H.	6½ ♂	Pneumonia; earache 3 wk.	Paracentesis, left	2-3 wk.	Coalescing; simple mastoidectomy	Hemolytic streptococcus	Uneventful; temperature normal 5th day	Uneventful	3 wk.	6 wk.	Normal	Slight depression; no prolapse; drum intact
18 S. J.	2½ ♀	Cold and earache 4 wk.	Paracentesis, left; spontaneous, right	4 wk.	Antrum sclerotic; coalescing tip; bilateral simple mastoidectomy	Hemolytic streptococcus	Uneventful; temperature normal 5th day	Uneventful	3 wk.	4 wk.	Normal	Moderate depression; no prolapse; drum intact
19 H. S.	7 ♀	Cold 2 wk.; earache 2 wk.	Paracentesis, left	2 wk.	Coalescing; extensive simple mastoidectomy	Hemolytic streptococcus	Uneventful; temperature normal 6th day	Uneventful	2½ wk.	5 wk.	Normal	Oval depression; no prolapse; drum intact
20 T. R.	58 ♀	Sore throat 2 wk.; earache 2 wk.	Paracentesis, left	2 wk.	Coalescing; extensive simple mastoidectomy	Hemolytic streptococcus	Stormy erysipelas; temperature normal 8th day	Uneventful	7 wk.	11 wk.	Poor, 50%	Moderate depression; moderate prolapse; drum intact
21 B. A.	42 ♂	Grip 1 mo.; earache 2 wk.	Spontaneous, left, 2 wk. before mastoidectomy	5½ wk.	Coalescing; extensive simple mastoidectomy	Pneumococcus	Uneventful; flat temperature	Uneventful	3 wk.	5 wk.	Normal	Moderate depression; no prolapse; drum intact
22 D. F.	4 ♂	Cold 3 wk.; earache 3 wk.	Paracentesis, left	3 wk.	Sclerotic antrum coalescing tip	Hemolytic streptococcus	Uneventful; temperature normal 3d day	Uneventful	2½ wk.	4 wk.	Normal	No depression; no prolapse; drum intact
23 R. L.	20 ♀	Cold 8 wk.; earache 8 wk.	Paracentesis, right, 3 times	9 wk.	Coalescing; extensive simple mastoidectomy	Nonhemolytic streptococcus	Uneventful; temperature normal 4th day	Uneventful	From onset	4 wk.	Normal	Slight depression; no prolapse; drum intact
24 L. T.	14 ♂	Cold 3 wk.; earache 2 wk.	Paracentesis, right and left	2 wk.	Coalescing; bilateral simple mastoidectomy	Hemolytic streptococcus	Uneventful; temperature normal 5th day	Uneventful	3 wk.	5 wk.	Normal	Slight depression; no prolapse; drum intact

In summing up the data obtained from the study of these lateral roentgenograms we were in agreement with Taylor,[6b] as quoted in the beginning of this paper, that in postoperative roentgenograms the operative fields are clearly visible, but we do feel that some residual cells often remain in the antral or the perilabyrinthian region and that there still may be successful healing. However, if more than these usual cells remain and if disease lies in the regions pointed out by Almour,[7] then delayed healing or nonhealing will make secondary operations necessary.

On further examining the postoperative roentgenograms, we noted that the middle ear canals were clear in all cases. There was no evidence of cell layers, and there appeared to be no regeneration of bone. In 2 of the cases in which mastoidectomy had been performed at an early age, new cells were visualized distal to the superior semicircular canal in the postoperative films taken years later. These cells, however, we feel, were due to developmental change, the mastoid growth proceeding from the infantile into the adult pneumatic mastoid, and not to regeneration of bone. The petrous apexes appeared normal in all cases.

In conclusion, it is my feeling that a sound philosophy must be adopted in dealing with mastoiditis. If one has a definite plan in mind and follows this routinely in all cases, he is less likely to fall amiss and find himself in trouble with most instead of only a few of his patients. It is better to wait a little longer before doing a mastoidectomy than to operate when the infection is in an early stage. If one follows a plan, he may expect a stormy postoperative course to be the exception, whereas if too early intervention has been attempted he may expect to find the postoperative course stormy in almost all cases.

I feel that preoperative roentgenograms are essential for the following reasons: (1) The type of mastoid can be foretold; (2) the density of the antral region can be noted; (3) the visibility and sharpness of outline of the lateral sinal wall can be detected; (4) the condition of the trabeculae and the cells can be observed, and finally (5) the appearance of the petrous apex can be examined. With this help the amount of operative work to be performed will be formulated, the possibility of a perisinal or a peridural abscess will be suspected, the extent of the breaking down of the trabeculae and the cells will be visualized and any involvement of the petrous apex will be determined.

Postoperative roentgenograms likewise, I feel, are essential. I advocate the making of roentgenograms immediately after operation, for comparison with the preoperative roentgenograms, and if at a subsequent time reoperation is considered, the comparison of roentgenograms taken at that time with the preoperative and the first postoperative pictures will be of great help. The more thorough the operation has

TABLE 2.—*Roentgenologic Study Before Operation*

Case	Patient	Date of Roentgenograms	Type of Mastoid	Auditory Canal	Antrum	Division into Cell Layers	Trabeculae	Cells	Sinus Plate	Operative Findings
1	A. J.	6/38	Small pneumatic	Clear	Not dense	Not present	Thick	Blurred	Visible; eroded at knee	Coalescing mastoid; perisinal abscess
2	S. P.	4/37	Pneumatic	Clear	Not dense	Combination	Absent, necrosed	Blurred	Visible; thick	Coalescing mastoid
3	S. P.	2/37	Small pneumatic	Clear	Not dense; cloudy	Not present	Absent; cells blurred	Blurred	Not visible	Coalescing mastoid
4	S. V.	3/35	Large pneumatic	Clear	Not dense	Large cells	Thin, indistinct	Blurred	Visible, thin	Coalescing mastoid
5	N. G.	11/33	Pneumatic	Occluded	Dense	Large cells	Thick, dense	Blurred	Not visible	Coalescing mastoid; pus granulations
6	G. A.	4/35	Large pneumatic	Occluded	Dense	Large cells	Thick in areas	Dense	Faint	Sclerosis; perisinal abscess
7	C. A.	3/37	Pneumatic	Occluded	Dense	Combination	Absent	Blurred, dense	Thin, eroded	Coalescing mastoid; sclerosis; perisinal abscess
8	H. M.	3/37	Pneumatic	Clear	Not dense	Combination	Thick	Blurred	Not visible; eroded ?	Coalescing mastoid; sinal wall intact
9	McG. E.	7/38	Pneumatic	Clear	Not dense	Combination	Thick	Blurred	Thick, visible	Coalescing mastoid; perisinal abscess
10	W. N.	10/33	Large pneumatic	Occluded	Dense	Double layer	Thick, hazy	Blurred, dense	Faint	Coalescing mastoid, bilateral
11	R. J.	7/38	Large pneumatic	Clear	Dense	Combination	Absent	Dense	Not visible; eroded ?	Coalescing mastoid; perisinal abscess; angle removed
12	S. A.	1/36	Large pneumatic	Slightly occluded	Dense	Double layer	Thick, hazy	Blurred, dense	Not visible	Coalescing mastoid
13	M. A.	10/38	Pneumatic	Occluded	Not dense; moderate	Double layer	Thin in areas	Blurred	Thick	Hemorrhagic mastoid
14	K. E.	9/37	Pneumatic	Occluded	Large, dense	Double layer	Thick, hazy	Blurred	Not visible	Coalescing mastoid, extensive
15	S. J.	4/37	Pneumatic	Clear	Not dense; large	Moderately large cells	Thick	Blurred	Visible	Coalescing mastoid; pus cavity
16	S. R.	2/35	Pneumatic	Occluded	Large, dense	Combination	Thick, hazy	Blurred	Not visible	Coalescing mastoid
17	M. H.	1/35	Pneumatic	Clear	Dense	Large cells	Thin	Blurred	Thin	Coalescing mastoid
18	S. J.	4/33	Infantile	Occluded	Not dense; cloudy	Small cells	Thick, hazy	Blurred, small	Not visible, left; visible, right	Sclerotic mastoid; pus necrosis; granulations
19	H. S.	1/38	Large pneumatic	Clear	Not dense; cloudy	Large cells	Thin, hazy	Blurred	Visible; thin	Coalescing mastoid
20	T. R.	5/36	Pneumatic	Cloudy	Dense	Combination	Thick	Blurred, dense	Not visible; eroded ?	Coalescing mastoid; cortex perforated
21	B. A.	4/36	Large pneumatic	Clear	Dense	Combination	Thin, hazy	Blurred	Slightly visible	Coalescing mastoid
22	D. F.	1/37	Small pneumatic	Clear	Not dense	Small cells	Thick, hazy	Blurred	Faint	Sclerotic mastoid; areas of granulation
23	R. L.	7/38	Pneumatic	Clear	Not dense	Moderately large cells	Thick, hazy	Blurred	Faint; visible	Coalescing mastoid; decalcified cortex with a spongy feel; granulations
24	L. T.	6/36	Pneumatic	Occluded	Not dense	Combination	Thick	Blurred, dense	Not visible	Coalescing mastoid (bilateral), early stage

been, the more clearly will the field be visualized, the more sharply the permanent structures will be outlined and the fewer residual cells will be evidenced. A comparison of these sets of roentgenograms will be of great aid in determining necessity and procedure in cases of secondary mastoid infection.

From our study of the postoperative roentgenograms of the patients chosen, we noted the following facts: (1) The defective area removed was in the contour of an acute, obtuse or right angled triangle; (2) the permanent structures were clearly and sharply outlined; (3) there were few, if any, remaining cells, and these were in the antral region, and (4) there was little or no roentgen evidence of regeneration of bone. It appeared that if the mastoid operation was performed on a young child there followed a developmental growth of cellular bone as the growth of the child progressed rather than a regeneration of new bone.

23	R. L.	7/38	Pneumatic	Clear	Not dense	Moderately large cells	Thick, hazy	Blurred	Faint; visible	Coalescing mastoid; decalcified cortex with a spongy feel; granulations
24	L. T.	6/36	Pneumatic	Occluded	Not dense	Combination	Thick	Blurred, dense	Not visible	Coalescing mastoid (bilateral), early stage

REPORT OF CASES

CASE 10.—W. N., a 6 year old school girl, when seen for the first time was acutely ill. The history revealed an infection of the upper respiratory tract of two weeks' duration, at the onset of which she had a spontaneous rupture of both ear drums. There was little or no discharge at any time from her ears. When seen she complained of headache and of pain in and behind both ears and showed evidence of postauricular edema and marked tenderness over both mastoids. There was some pus in both external meatuses. Otherwise the results of physical examination were negative.

A diagnosis of bilateral mastoiditis was made, and the patient was admitted to the hospital. Her temperature was 103 F., the pulse rate 94 and the respiratory rate 34. Her blood count showed 68 per cent hemoglobin content, 3,620,000 red blood cells and 12,500 white blood cells, with 62 per cent polymorphonuclears and 6 band forms. Her urine showed acetone and albumin (a trace). Roentgenograms revealed cloudiness and loss of detail of both mastoids, greater on the left side, and there was a marked increase of density in the antral region.

At the operation both mastoids were seen to be of the coalescing type, there was pus under pressure but no evidence of perisinal abscess, and granulations and dead bone were present throughout. An extensive simple bilateral mastoidectomy was performed. Culture of material obtained at operation yielded hemolytic streptococcus.

The patient made an uneventful recovery; her temperature became normal on the third postoperative day and she was allowed to go home on the seventh day. Both middle ears dried by the end of two and one-half weeks, and there was complete healing in five weeks.

At the end of the sixth week the child had an attack of dizziness, nausea and vomiting, associated with a rotary nystagmus to the right. She was put to bed, watched carefully and kept there for one month, during which time all symptoms subsided. From that time she made an uneventful recovery and today continues to be in perfect health. Her mastoidectomy incisions healed with flat scars and moderate depressions and there was only a slight prolapse of her external auditory canals. Her ear drums are intact, and hearing is normal in both ears.

TABLE 3.—*Roentgenologic Study After Operation*

Case; Patient	Date of Roentgenograms	Interval Between Preoperative and Postoperative Roentgenograms	Type of Mastoid	Defect	Canal	Antrum	Cell Layers	Trabeculae	Cells	Sinus Plate; Sinodural Angle	Regeneration of Bone	Apex
1 A. J.	5/39	11 mo.	Small pneumatic	Acute angle triangle	Clear position?	Clear	Removed	Absent	Few; proximal to labyrinth, hazy	Absent Absent	None	Normal
2 S. P.	3/39	1 yr., 11 mo.	Pneumatic	Acute angle triangle	Clear	Clear	Removed	Absent	Absent; bone hazy	Visible Present	None	Normal
3 S. P.	4/39	2 yr., 2 mo.	Infantile; small pneumatic	Right angle triangle	Very clear	Clear	Removed	Absent	Distal to labyrinth, clear	Visible, thin Present, thin	Cells developed distal to labyrinth	Normal
4 S. V.	4/39	4 yr., 1 mo.	Large pneumatic	Right angle triangle	Clear	Clear; few cells	Removed	Hazy	Clear, periantral	Present Present	None	Normal
5 N. G.	4/39	5 yr., 5 mo.	Pneumatic	Acute angle triangle	Small; clear	Clear	Removed	Absent	Few, periantral, clear	Present Present	None	Normal
6 G. A.	5/39	4 yr., 1 mo.	Large pneumatic	Right angle triangle	Clear	Hazy	Removed	Thick	Few, clear, proximal to labyrinth	Thin Present	None	Normal
7 C. A.	5/39	2 yr., 2 mo.	Pneumatic	Right angle triangle	Clear position?	Hazy	Removed	Thin	Clear, periantral, few	Thick Present	None	Normal
8 H. M.	5/39	2 yr., 2 mo.	Pneumatic	Right angle triangle	Clear	Clear	Removed	Absent	Absent	Present Present, thick	None	Normal
9 McG. E.	5/39	10 mo.	Pneumatic	Right angle triangle	Clear	Clear	Removed	Thin	Few, hazy, proximal to labyrinth	Thin Present	None	Normal
10 W. N.	6/39	5 yr., 8 mo.	Large pneumatic	Right angle triangle	Clear	Clear	Removed	Thin	Clear, proximal to labyrinth (both sides)	Thin Present (both sides)	None	Normal
11 R. J.	6/39	11 mo.	Large pneumatic	Acute angle triangle	Clear	Clear	Removed	Thin	Few, deep, proximal to labyrinth	Absent Absent	None	Normal
12 S. A.	4/39	3 yr., 3 mo.	Large pneumatic	Obtuse angle triangle	Clear	Dense	Removed	Thick	Dense, proximal to labyrinth	Absent Present	None	Normal
13 M. A.	5/39	7 mo.	Pneumatic	Right angle triangle	Clear	Clear	Removed	Absent	Absent	Thin Present	None	Normal
14 K. E.	6/39	1 yr., 9 mo.	Pneumatic	Obtuse angle triangle	Clear position?	Clear	Removed	Absent	Few, clear, proximal to labyrinth	Thin Present	None	Normal
15 S. J.	6/39	2 yr., 2 mo.	Pneumatic	Right angle triangle	Clear	Clear	Removed	Absent	Few, proximal to labyrinth, clear	Thin Present	None	Normal
16 S. R.	6/39	4 yr., 4 mo.	Pneumatic	Obtuse angle triangle	Small; clear	Clear; few cells	Removed	Thin	Few, periantral	Thin Present	None	Normal
17 M. H.	6/39	4 yr., 5 mo.	Pneumatic	Right angle triangle	Clear	Clear	Removed	Thick	Few, periantral, proximal to labyrinth	Thin Present	None	Normal
18 S. J.	6/39	6 yr., 2 mo.	Infantile	Right angle triangle (both)	Clear	Cloudy	Removed	Thick	Many, periantral; deep, subantral	Thick Present	None; cells developed with age	Normal
19 H. S.	6/39	1 yr., 5 mo.	Large pneumatic	Right angle triangle	Clear	Clear	Removed	Absent	Few, clear, proximal to labyrinth	Thin Present	None	Normal
20 T. R.	7/39	3 yr., 2 mo.	Pneumatic	Obtuse angle triangle	Clear	Hazy	Removed	Thick	Few, periantral	Thin Present	None	Normal
21 B. A.	8/39	3 yr., 4 mo.	Pneumatic	Right angle triangle	Clear	Clear	Removed	Thin	Few, proximal to labyrinth	Thin Present	None	Normal
22 D. F.	7/39	2 yr., 6 mo.	Pneumatic	Right angle triangle	Clear	Clear	Removed	Thin	Few, periantral	Thin Present	None	Normal
[illegible]	7/39	1 yr.	Pneumatic	Obtuse angle	Clear	Hazy	Removed	Absent	Absent	[illegible]	[illegible]	Normal

CASE 11.—R. J., a 5½ year old boy, had had repeated colds and earaches for three months. A paracentesis had been performed in the left side seven weeks before his present illness, after which infection subsided in a few days. He was seen again ten days previous to hospitalization; at this time he had a reinfection of the left ear drum. Paracentesis was performed, and there followed a profuse purulent discharge from the ear. His symptoms became progressively worse, and he was finally hospitalized and roentgenograms of the ear were made. Definite evidence of a pathologic condition in the left mastoid, with cloudiness of the trabeculae, particularly posteriorly over the sinus plate, was reported.

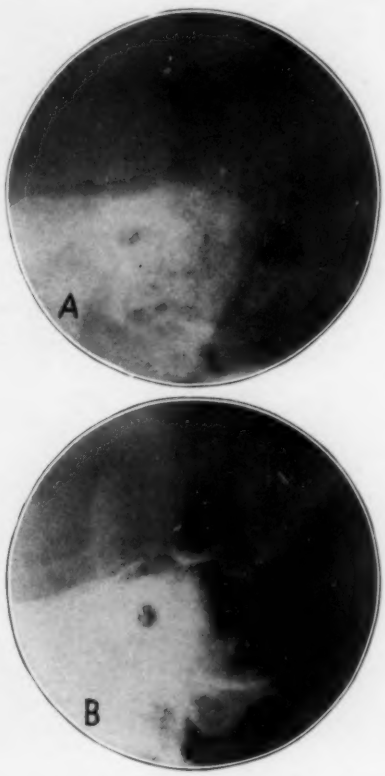

Fig. 1.—Roentgenograms of an extensively involved mastoid bone: *A*, preoperative, showing dense antral region; *B*, postoperative, showing thoroughly cleaned out mastoid. Notice the sharpness of detail of the permanent structures and the few remaining cells.

At operation an extensively coalescing mastoid was found. There was a perisinal abscess, with granulations on the sinal wall, extending beneath the angle of the superior petrosal sinus and involving the dura. An extensive simple mastoidectomy was performed, and the bony angle of the superior petrosal sinus was removed, freeing the granulations from all surrounding bone. The culture obtained was an undifferentiated pneumococcus.

The patient made an uneventful postoperative recovery. He was discharged on the seventh postoperative day, his middle ear was dry by the end of two and

one-half weeks and the posterior wound was entirely healed in four weeks. His scar was flat, with little depression, in spite of the removal of the angle. There was no prolapse of the canal, and the drum was intact. His hearing was normal.

This case illustrates the possibility that a perisinal abscess may be present and not demonstrated by roentgenograms. The infection undoubtedly was of long duration, probably beginning before the first paracentesis; the evidence was borne out not only by the operative findings but also by the report of the culture of pneumococcus, a treacherous organism, which acts silently, giving rise to few symptoms.

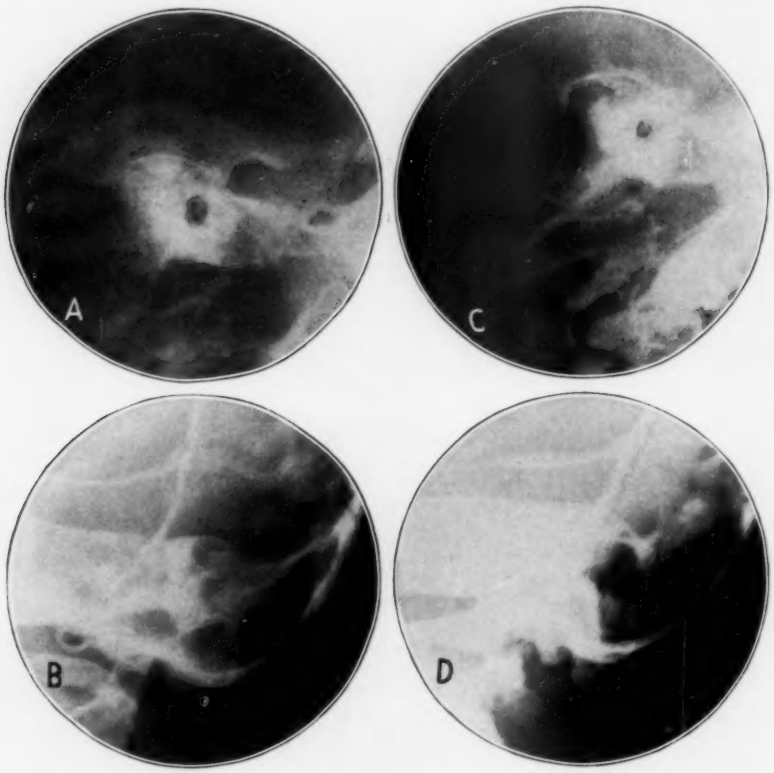

Fig. 2.—Roentgenograms of a patient: *A*, preoperative lateral view, showing dense antral region but no evidence of perisinal abscess; *B*, preoperative anterior-posterior view; *C*, postoperative lateral view, showing the result of the removal of the angle of the superior petrosal sinus; *D*, anterior-posterior view, showing the postoperative triangular defect resulting from mastoidectomy.

Case 13.—M. A., a 5 year old white girl, when seen for the first time was acutely ill, with a temperature of 104 F. and complained of pain in her left ear of three days' duration, which was associated with a spontaneous rupture of her ear drum and with a progressive increase in tenderness and swelling behind her left ear.

Her past history was entirely irrelevant with the exception of a slight head cold one week previously, during which illness she continued to attend school.

A diagnosis of mastoiditis on the left side was made, and she was immediately hospitalized. On admission the laboratory reported the hemoglobin content 87 per cent and the blood count 4,760,000 red blood cells and 13,200 white blood cells, with 66 per cent polymorphonuclears. Her urine was normal. The roentgenogram revealed an acute infection of the left mastoid, showing the mastoid to be of the pneumatic type, with thinning of the trabeculae and blurring of the cells, accentuation of the anterior sinal wall and general cloudiness of the mastoid bone.

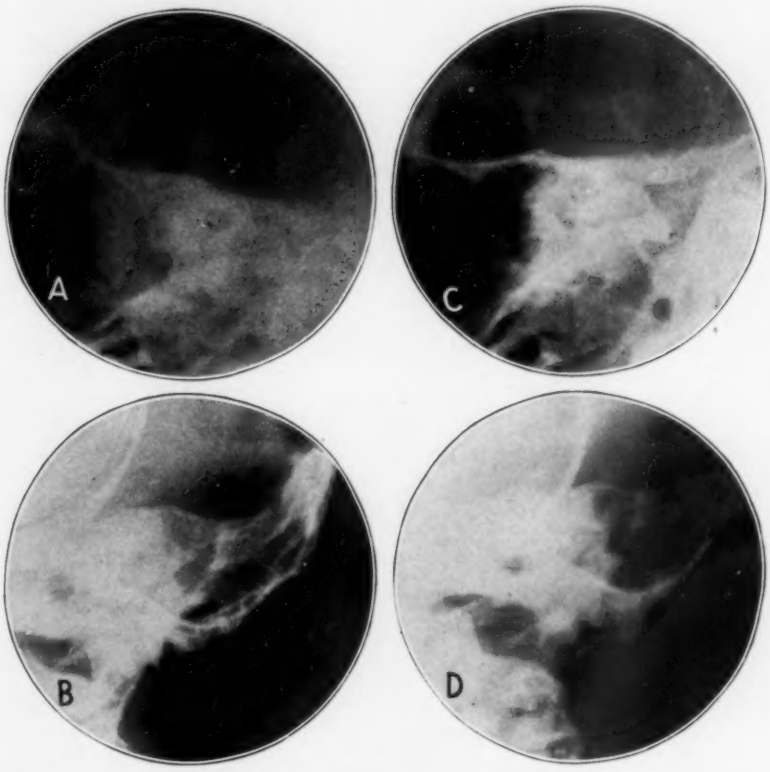

Fig. 3.—Roentgenograms of a patient with a hemorrhagic mastoid: *A*, preoperative lateral view, showing early extensive involvement of the bone; *B*, preoperative anterior-posterior view; *C*, postoperative lateral view, showing the completeness of the removal of the mastoid cells; *D*, postoperative anterior-posterior view.

The following morning an extensive simple mastoidectomy was performed. A hemorrhagic mastoid with little evidence of free pus but with trabeculae extensively diseased was revealed. The bacteriologic report was hemolytic streptococcus.

The patient's recovery after operation was uneventful; the temperature became normal by lysis, and on the sixth day she was ready for discharge to her home. At home her recovery was complete in five weeks, the middle ear being dry in two and one-half weeks. The posterior wound healed with a flat scar and little

depression, the ear drum was normal and there was only slight prolapse of the aural canal. The child had no further involvement.

The case is presented as one of a rather infrequent, fulminating, hemorrhagic type of mastoiditis, with operation early in the course of the disease and with an uneventful postoperative course.

CASE 18.—S. J., a 2 year old girl, had a history of repeated otitis and repeated bilateral paracentesis over a two month period after an acute infection of the

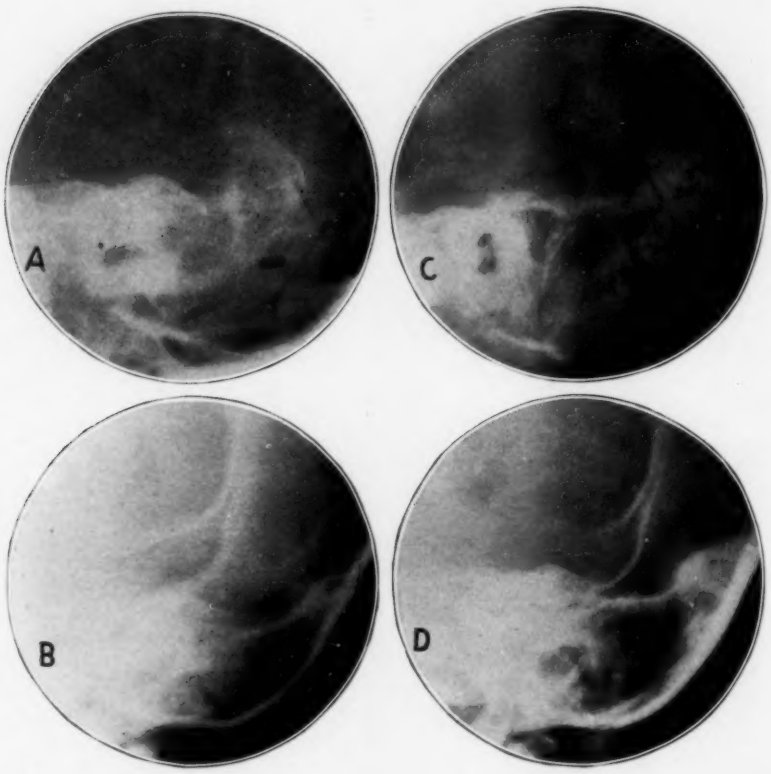

Fig. 4.—Roentgenograms of an infantile mastoid: *A*, lateral preoperative view; *B*, anterior-posterior preoperative view; *C*, lateral postoperative view, showing developmental growth of bone to adult stage six years after mastoidectomy; *D*, anterior-posterior postoperative view.

upper respiratory tract. Her mother stated that she had been restless at night and had been crying with pain and that her temperature had been fluctuating for the past several weeks.

When she was examined, both ears were discharging, and there was acute tenderness over both mastoid bones. Her temperature was 102 F., the pulse rate 120 and the respiratory rate 24. She was hospitalized, and the roentgenographic report showed both of her mastoids to be of an infantile type, with haziness and loss of detail.

An extensive simple mastoidectomy was performed on each side. The mastoids were of the undeveloped type, with areas of sclerosis throughout and areas of necrosis, free pus and granulations.

The child made an uneventful postoperative recovery; the temperature became normal on the fourth day, and she was allowed to go home on the sixth day. At home an uneventful course followed. The middle ears became dry by the third week, and complete healing had occurred by the fifth week. Her operative scars were flat, only slight depressions were noted, and there was no prolapse of her aural canals; her ear drums were intact and her hearing normal.

This case is presented to show roentgenograms of an infantile type of mastoid, which, when roentgenograms were made six years later, showed growth of bone with development into an adult type of mastoid. There appeared in the roentgenogram of the mastoid area a developmental growth of bone to the adult stage rather than a regeneration of bone.

Drs. Charles F. Baker and W. James Marquis reviewed the roentgenograms and the subject matter used in the preparation of this paper.

223 Roseville Avenue.

IDIOPATHIC HYPEROSTOSIS OF THE SKULL

FRANCIS L. LEDERER, M.D.
CHICAGO

The osseous structures of the head are capable of undergoing pathologic changes which are of interest to the otolaryngologist. The bony labyrinth of the ear, for example, may be affected by such diseases as fragilitas ossium, osteitis deformans, osteoarthritis, osteogenesis imperfecta, osteomalacia, osteopsathyrosis, rickets, Paget's disease, senile osteoporosis, chondrodystrophy and athyreopituitary dwarfism. Many of these may affect any and all parts of the skull. There is a group of idiopathic hyperostoses of the skull, which have long been considered rare, viz., osteitis deformans, or Paget's disease, and craniosclerosis, or leontiasis ossea. Recently Coppo,[1] in connection with a description of a patient with the latter condition, reviewed the literature extensively and found the cases recorded not numerous.

Virchow [2] proposed the term "leontiasis ossea" for a particular form of hyperostosis of the skull. The name was suggested by the appearance which the face of the patient came to assume, due to enlargement in the region of the superior maxilla combined with deepening of the orbital cavities, which produced a picture similar to that of leonine leprosy. The term itself, therefore, was descriptive only of the appearance of the patient, not of the underlying pathologic changes. As a result, many conditions of dissimilar bone changes have been grouped under the heading "leontiasis ossea" simply because they are associated with facial asymmetry.

ETIOLOGY

Opinions regarding the etiologic factors involved in leontiasis ossea are at variance. Some authors have mentioned that the disease is a form of rickets (craniosclerosis rachitica); some have expressed the belief that it results from infections such as phlegmons and abscesses of the skin, erysipelas, dental abscesses and abscesses of the lacrimal sac, while others have considered it to be due to an osseous dysplasia resulting from trauma or infection, in which osteogenic activity is materially affected. The possible relation to endocrine dysfunction has been frequently sug-

From the Department of Otology, Rhinology and Laryngology of the University of Illinois College of Medicine.

1. Coppo, E.: Sindrome nasale nella leontiasi craniofaciale, Valsalva **16**:76, 1938.

2. Virchow, R.: Die krankhaften Geschwülste, Berlin, A. Hirschwald, 1864, vol. 2, p. 23.

gested. While the condition has been likened to acromegaly, it differs in that the changes in so-called leontiasis ossea are limited to the osseous tissue, whereas the former disease affects osseous tissue and the soft parts as well. The role of syphilis as a factor has also been stressed by a number of contributors to the literature on this entity.

PATHOLOGIC ANATOMY

The similarity from the standpoint of comparative pathologic anatomy between leontiasis ossea and Paget's disease has frequently been emphasized by these authors, and both diseases have been referred to as "forms of idiopathic hyperostosis" without any attempt to make a sharp distinction between them. The main differences offered have been that leontiasis ossea is a form of cured osteitis deformans and that it is a form of localized osteitis fibrosa or a type of circumscribed osteosclerosis. Paget himself established a clear distinction between the two forms. Paget's disease has been pointed out as a disease of advanced age (after the fifth decade), in which there is no antecedent history of syphilis, tuberculosis or neoplasms, which is seldom localized to the head and which rarely produces a disfigurement of the facial contour.

Coppo accepted Monti's classification of leontiasis ossea, compiled as the result of a study of the cases recorded in the literature:

(1) *Leontiasis ossea facialis autonoma:* This constitutes the true type of the disease, affecting the young, represented by a slight productive periosteal change, beginning in the superior maxilla, limited in localization to the bones of the face and characterized by an extremely insidious progress. Histologically there is eburnation of the compact portion and of the spongy bone due to a disappearance of the osseous medulla. The nutrient vessels maintain their activity in nourishing the new osteoblasts and osteocytes. In this form of the disease there is a definite lack of a corresponding activity of bony absorption.

(2) *Leontiasis ossea syphilitica:* This type differs from the preceding form in that it extends to involve the entire skull very early in the disease. Inasmuch as this form also affects the very young, symptoms of craniostenosis are frequent.

(3) *Leontiasis ossea craniofacialis of osteodystrophia fibrosa:* More commonly this form, also occurring in the young, affects the interior of the skull, but it may also produce the characteristic external deformity of the disease. It differs essentially from the other forms in that the process begins in the osseous medulla, which becomes fibrous, while the lacunas become resorbed and the superficial compact zone becomes localized where the osteoclasts are present in large numbers.

(4) *Leontiasis ossea of osteitis deformans:* In this type the deformity results from a rarifying osteitis and a process of condensation, which alternate. The latter is predominant and leads to an osseous hyperostosis, which is characterized by an intense irregularity of the disposition of the lamellar system.

SYMPTOMATOLOGY

The osseous changes occur so insidiously that early recognition based on the symptom complex is difficult. Essentially there may be external deformity, nasal obstruction, mucopurulent nasal discharge, headaches, alterations in the voice (rhinolalia clausa) and occasionally ocular disturbances, such as exophthalmos, visual difficulties and lacrimation. With further encroachment on vital structures, and when symptoms of craniostenosis prevail, there may be evidence of cortical involvement in association with compression of vessels and nerves. There may be intense neuralgias, diminution of vision, which may result in complete amaurosis, olfactory and auditory disturbances, paralyses, convulsions, contractures and the like. The clinical picture is not so intense in some cases, because of the slow development of the disease. In such cases death ensues in later years because of some intercurrent disease.

ROENTGEN EVIDENCE

Roentgenographic studies are of importance in recognizing this condition and determining the localization of the process. According to Mayer,[3] there are a noticeable raised thickening and an intense and uniform eburnation of the bone. The superficial contour of the diseased bone is sharply outlined and appears smooth. The condition usually limits itself to the anatomic divisions of the affected bones of the skull. It is most commonly localized to the mandible or to the maxilla. In Mayer's experience, the disease seldom affects the other bones of the skull, and when it does, it is differentiated from the common forms of hyperostosis with considerable difficulty. It is of importance to compare the roentgenographic appearance of the skull altered by so-called leontiasis ossea with that of the skull affected with Paget's disease, in which the sharp outline, definite localization and lack of uniform density are conspicuously absent.

PROGNOSIS

The prognosis of leontiasis ossea is favorable in those cases in which the progress of the disease has been slow, but it is grave in the instances in which the symptoms of craniostenosis supervene. Death, however, ensues as the result of intercurrent infections, in the majority of cases.

TREATMENT

Therapy is neither specific nor effective in instances of extensive involvement. Pain is controlled by sedation and, when possible, neural injection or evulsion. In cases in which there is syphilis, antisyphilitic therapy is instituted without the expectation of regression of the osteo-

3. Mayer, E. G.: Ueber Röntgenbefund bei retrobulbären Erkrankungen unklarer Aetiologie und ihre differentialdiagnostische Wertung, Acta radiol. **9**:383, 1928; personal communication to the author, March 1938.

genic changes. Coppo stated the belief that resection of anatomic parts is worth while and effective in those cases in which pain does not respond to medical treatment. He reported in great detail, both clinically and surgically, such an instance in which he resected the right superior maxilla. Incidentally, the patient had a history of a dental abscess, frequent trauma, a positive Wassermann reaction and increased blood calcium.

Fig. 1.—View from the right side of the skull.

REPORT OF A CASE

The Department of Otology, Rhinology and Laryngology of the University of Illinois College of Medicine has been fortunate for many years in receiving all castoff cadaver material after the students have finished their dissections. This material has furnished excellent opportunities for anatomic studies and the collection of permanent museum specimens. Among these specimens was a head of unusual proportions and contour. Tracing of the records revealed that the patient, a man 50 years of age, died of pneumonia in one of the municipal institutions. As the patient entered the hospital in a critical condition, no past history is available, nor was there in the clinical records any reference to the deformity of the face. There were no gross osseous changes in any other part of the body.

The opportunity to study the entire skull minutely is unusual, if one may judge by the medical literature, and for this reason a detailed description is given.

The squamous portion of the frontal bone was entirely sclerotic but only slightly thickened. The frontal sinus on the left side was present but was reduced to the size of a pea, and there was no communication to the nose. The foramen caecum was present and of normal size. The right frontal sinus was not opened but appeared slitlike (fig. 3). The crista galli was slightly thickened. The lamina cribrosa on the left side was present but narrow. The roof of the ethmoid sinus and the orbit was much thickened and sclerotic. The lamina cribrosa on the right side was absent. The sella turcica was reduced in size in its sagittal

Fig. 2.—Front view of the skull, showing the enormous increase in the size of the right mandible.

diameter. This narrowing was produced by a thickening of the anterior wall, which projected into the sella turcica. The posterior wall and the dorsum sellae were normal. The optic foramen on the left was present but also narrowed. The right optic foramen was likewise encroached on. The superior orbital fissure on the right side was narrowed laterally. The left fissure was not altered.

Both middle fossae were normal. The squamous portion of the temporal bone was normal, revealing a lamina interna, a lamina externa and diploe. The right petrous pyramid was not unusual, with the exception of the internal auditory meatus, which was very narrow. On the left side the pyramid had been partly disturbed in its preparation but revealed a diploic tip and a very narrow internal auditory meatus. The bony inner ear, the jugular bulb and the eustachian tube were normal.

Only the right posterior fossa was normal throughout. The squamous portion of the occipital bone was without change, but the foramen magnum was slightly narrowed. This narrowing was produced by thickening and sclerosis of the clivus. The atlas was normal, but the clivus of the epistropheus was thickened and sclerotic.

Viewing the nose from behind, one saw enormous thickening of the bony septum, which almost obliterated both nasal cavities. The turbinates on the right side appeared to be atrophic, apparently because of pressure. On the left side, the posterior ends of the middle and inferior turbinates were present and likewise

Fig. 3.—Right cut surface of the skull.

appeared atrophic. The roof of the epipharynx was thickened and almost entirely obliterated. The lateral walls of the epipharynx were apparently normal.

The skull was divided by a sagittal section, which began to the right of the crista galli and succeeded in separating the nasal septum and the hard palate into a broader left and a narrower right portion. The left cut surface showed that the entire bony septum had apparently changed into a thick sclerotic osseous mass. The margins between the lamina perpendicularis and the vomer were obliterated. The perpendicular plate extended downward to such a degree that it encroached on the quadrangular cartilage, shortening the vertical diameter of the latter. The perpendicular plate was attached to the left nasal bone. There were enormous thickening and sclerosis of the entire hard palate, most marked

in the anterior part. Between the hard palate and vomer, the sphenoid process of the quadrangular cartilage was present. The sphenoid sinus was entirely obliterated.

The right cut surface (fig. 3) showed the previously mentioned thickening and sclerosis of the frontal squama and almost complete absence of the frontal sinus. The nasal bone was normal. The anterior ethmoidal foramen was also normal. The septum and the previously described changes were readily seen. On the lateral wall of the nose the inferior turbinate was found to be compressed in its anterior half by the thickened hard palate and markedly impinged on its posterior half by the thickened vomer. The anterior portion of the middle turbinate was seen and was just slightly compressed on its medial surface. The anterior part of the middle meatus was observed. The mandible was seen to be enormously thickened.

Microscopic Studies.—Microscopic examination of a cross section of the greater wing of the sphenoid bone showed an absence of the normal osseous structure. On the site of the outer and inner tables, there was not a continuous layer of bone but small haversian spaces with concentric lamellar bone. These formed haversian systems consisted of new outer and inner layers of the bone. From these newly formed layers a framework of bone branched off, which included more or less wide marrow spaces. In these marrow spaces there was loose connective tissue, but signs of apposition or of destruction of bone were not to be seen at the margins of these spaces. This finding is not entirely reliable, as the skull was macerated for some time before microscopic examination was possible. In many places islands of compact bone were found in the region of the diploe, consisting of well arranged haversian lamellae surrounding very narrow haversian canals. As far as the study of the specimen permits, it can be said that there was a marked tendency toward reconstruction of the diploe as well as of both compact layers of the bone, characterized by replacement of both compact layers, internal and external, by new compact layers and by an eburnation of the regional diploe.

COMMENT AND SUMMARY

The described specimen presents a classic example of leontiasis ossea faciei as first described by Virchow. However, this diagnosis needs further elucidation inasmuch as "leontiasis ossea faciei" is a more or less collective term for numerous diseases which produce considerable deformity of the face but which differ in their clinical course and prognosis. Consequently, it is necessary to attempt an exact diagnosis from the study of the specimen. This is somewhat difficult, as the specimen is a museum specimen and exact data concerning the patient are lacking. Therefore, more cannot be expected than to make a diagnosis with the most probability possible.

Monti classified four diseases which may lead to definite deformity of the face and of the skull: 1. Leontiasis ossea facialis autonoma, which is the true leontiasis, beginning in early youth as productive periostitis of slight degree. The disease is slowly progressive and affects only the bones of the face. Microscopically there is a disappearance of marrow with subsequent eburnation and thickening of the bone. Monti expressed the belief that this disease is due to lues congenita tarda. 2. Leontiasis ossea syphilitica. a disease which differs from leontiasis ossea facialis

autonoma in that it spreads rapidly over the entire skull and produces early craniostenosis. 3. Leontiasis ossea craniofacialis (fibrous osteodystrophy), also a disease which sets in in youth and which seems to be identical with the osteitis fibrosa of Recklinghausen. 4. Leontiasis ossea of the cranium, of the type of Paget, a disease which affects persons of the older age groups (after the fiftieth year of life) and involves mainly the lower extremities, seldom the skull. In the latter case the vault and the base of the skull are more often affected than the face.

It seems that Monti overestimated the importance of syphilis as a cause of the leontiasis, while he apparently underestimated the importance of the roentgen examination for the differential diagnosis of that disease. Consequently, the description given recently by Kienböck [4] seems of greater clinical importance. Kienböck mentioned the following diseases which might lead to deformity of the face: (1) osteitis fibrosa of Recklinghausen; (2) osteitis deformans of Paget; (3) true leontiasis ossea faciei of Virchow; (4) tumors of the bone (osteoma, chondroma and sarcoma); (5) lymphogranulomatosis, and (6) acromegaly.

Among these conditions, the diagnosis of the diseases mentioned as 4, 5 and 6 is not difficult. Much more difficult is the differential diagnosis between osteitis fibrosa, osteitis deformans and true leontiasis. It must be emphasized that leontiasis ossea is far more infrequent in occurrence than osteitis fibrosa or the more frequently observed osteitis deformans. Subjective complaints are in general moderate in all three diseases; they also may be entirely lacking. As far as the clinical course is concerned, it seems that leontiasis ossea has its onset in childhood or in early youth. As a matter of fact, Mayer has observed the disease frequently in infants and children but never in older persons. Toward the end of puberty the thickening of the bone ceases to progress or, at most, goes on for a few more years. Rarely is the disease progressive during the entire span of life, leading to extreme deformity, general pallor and stenosis in the areas of the nose, of the ear and also of the eye. Osteitis fibrosa also sets in early in life, but occasionally also in adult life, and is progressive over a long period. Osteitis deformans sets in at the earliest in the sixteenth or eighteenth year of life but in the majority of cases usually begins later, i. e., from the fortieth to the sixtieth year, and continues to progress.

Osteitis deformans, osteitis fibrosa and leontiasis ossea lead to enlargements of the facial contour which are, in general, similar in all three conditions. As to the arrangement of these thickenings, there is unilateral, or occasionally bilateral, symmetric enlargement of the face. Furthermore, either one or more bones may be affected; rarely are all of the bones of the face involved. Most frequently affected are the superior

4. Kienböck, R.: Leontiasis ossea faciei Virchows, Beitr. z. klin. Chir. **171**: 25, 1940.

maxilla, the malar bone, the ethmoid and the nasal bones. According to Mayer, the sphenoid bone, the parietal bones and, finally, the mandible may also be involved. However, there is one point which distinguishes leontiasis ossea definitely from Paget's and from Recklinghausen's disease. While in the latter disease occasionally only the skull and more often also the other parts of the skeleton are affected (symptomatic leontiasis), in true leontiasis ossea the skull solely is affected.

At first, the described hyperostoses of the bone do not produce visible deformity of the face. However, when the disease progresses, one finds diffuse enlargement of the face, which finally leads to definite deformity due to bony projections and humps. In this stage, obstruction of the nose, thickening of the nasal turbinates, diminution of hearing, tinnitus, exophthalmos and visual disturbances are noticed. In leontiasis ossea the teeth separate and are diagonal, but they do not fall out at all or do so very late. In osteitis fibrosa, the teeth soon fall out, particularly in those sites of the maxilla which are affected by the disease. In osteitis deformans, all of the teeth fall out early.

Roentgenologic studies in cases of leontiasis ossea evidence a great and uniform thickening of the bone. In roentgenograms made in cases of Paget's disease such areas appear somewhat foggy, spotty and dense; seldom do they appear definitely sclerosed. In cases of Recklinghausen's disease two types of change have been shown, viz., the destruction of bone produced by the development of cysts and the alteration produced by expansion of the bony cortex and later by the diffuse sclerosis. As far as leontiasis ossea particularly is concerned (and I identify this condition as idiopathic sclerosing hyperostosis), the most characteristic roentgen signs, according to Mayer, are the following: 1. There are considerable thickening and increased density of the bone, the thickening being intense and uniform. 2. The surfaces of the affected bone are entirely smooth and sharply defined. 3. The disease does not go beyond the anatomic margins of the bone or of a part of the bone if the latter has developed from one or more bony "anlagen." Kienböck emphasized the fact that one gets the impression that the disease starts in one part of the skeleton—for instance, in the superior maxilla—and extends slowly into the surroundings. The thickening of the bone takes place mainly in the direction of the least resistance: in the superior maxilla, toward the surface of the cheek and on the chin and the forehead. However, there are also changes in the walls of the sinuses. A less marked thickening takes place in other directions, particularly toward the mouth and the cranial cavity. In no other place is there a softening or a deformity of the bone due to malacia. Particularly is there no elevation of the base of the skull, which is so significant of Paget's disease.

The cause of leontiasis ossea, as well as that of osteitis deformans, is not known, while in certain forms of osteitis fibrosa an association of the

disease of the bones with disturbed function of the parathyroid glands is probable. Accordingly, the treatment is symptomatic and consists of removal of the bony enlargements in the nose in order to procure a free breathing space. In cases of osteitis fibrosa, according to Kienböck, one must search for a tumor of the parathyroid glands when there is generalized osteitis fibrosa, when the development of cysts in the skeleton is demonstrable, when hyperglycemia is found, when the patient is more than 13 years old and particularly when there is a palpable enlargement of the thyroid gland.

Considering my case on the basis of the aforementioned descriptions, I can exclude with certainty true tumor of the bones, lymphogranuloma and acromegaly. Consequently, only the differential diagnosis between osteitis fibrosa, osteitis deformans and true leontiasis ossea comes into consideration. But this differential diagnosis also can be simplified, because, in spite of the age of the patient, the diagnosis of Paget's disease can be excluded for the following reasons: 1. In the specimen examined, the vault of the skull was not affected, as it always is in advanced stages of Paget's disease. 2. There was no elevation of the base of the skull, such as is frequently found in cases of Paget's disease. 3. The other parts of the skeleton were not affected in the specimen examined, while they are usually involved in Paget's disease. 4. In the roentgenogram, the thickened bones of the specimen showed definite sclerotic changes, while in Paget's disease the thickened bones evidence the well known spotty and spongy structures. 5. The microscopic examination of the thickened bone of the specimen did not show the well known mosaic structures (Schmorl) which are to be seen with Paget's disease, even in exhumed bones. These reasons are sufficient to exclude the diagnosis of Paget's disease in my case.

Consequently, I am concerned only with the differential diagnosis between leontiasis ossea and osteitis fibrosa. However, since in this case the other parts of the skeleton were normal, as has been mentioned before, only a localized form of osteitis fibrosa comes into consideration. In considering the differential diagnosis, I have decided in favor of leontiasis ossea for the following reasons: 1. The bony thickenings showed definitely the characteristic behavior of leontiasis ossea. For example, there were intense and uniform enlargements and bony densities which grew in the direction of the least resistance, to the outside or into the different sinuses of the skull, whereas the mouth and the cavity of the skull were spared. The surfaces of the thickened bone were smooth, and the enlargements did not go beyond the anatomic margins of the bones. Softenings of the bone and cysts, the latter so significant of the localized form of osteitis fibrosa, were absent in the specimen. 2. While in the localized form of osteitis fibrosa the teeth soon fall out in the affected areas, in the specimen the teeth were present. Consequently, I feel

justified in making the final diagnosis of leontiasis ossea faciei of Virchow. Inasmuch as this disease is relatively rare and seldom is it that one has an opportunity to obtain a complete anatomic specimen of the skull affected by the disease, I felt it opportune to describe the case in extenso.

For the rhinologist, it is mainly of interest to study the condition of the paranasal sinuses in the presence of these diseases. Mayer (quoted by Brunner [5]) examined 24 roentgenograms of skulls affected by Paget's disease. Among the 24 cases represented by these roentgenograms, there were 15 (62.5 per cent) in which the paranasal sinuses were normal; in 2 cases there was excessive development of the paranasal sinuses, particularly of the frontal sinuses; in 5 cases the sinuses were definitely affected, and in 2 cases they could not be found at all. Consequently, we must say that in cases of Paget's disease an obliteration of one or more of the paranasal sinuses may occur. Brunner and Grabscheid have demonstrated by microscopic examinations of skulls affected by Paget's disease the manner in which obliteration of the sinuses occurs.

There is, of course, no similar examination at our disposal so far as osteitis fibrosa or leontiasis ossea are concerned, since, as mentioned previously, these diseases are of far more infrequent occurrence than is Paget's disease. However, the case presented shows that also in leontiasis ossea obliteration of the sinuses may occur. Of especial interest is the finding in this case that the nasal septum has undergone excessive thickening, whereas the nasal turbinates have atrophied as a result of pressure from the nasal septum. This finding is interesting because usually in leontiasis ossea the turbinates become enlarged and the nasal septum becomes thickened. It was impossible, of course, to make a microscopic study in order to demonstrate the development of the thickenings in this case of leontiasis ossea, inasmuch as the disease was greatly advanced when the patient died and the specimen was not in condition to permit more extensive studies.

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5. Brunner, H.: Die Nebenhöhlen der Nase bei Osteitis deformans (Paget) des Schädels, Acta oto-laryng. **26**:174, 1938.

CHRONIC MAXILLARY SINUSITIS

REPORT OF SEVENTY CASES IN WHICH RESORT WAS MADE TO THE CALDWELL-LUC OPERATION

J. B. NAIL, M.D.
Head of the Department of Otolaryngology, Wichita Falls Clinic Hospital
WICHITA FALLS, TEXAS

The Caldwell-Luc operation, introduced in 1897, has continued to be one of the most important surgical procedures for the treatment of chronic infection of the maxillary sinus.[1] Variations have been made in the technic, but the fundamental principles remain the same. The history of the development of this operation, which is familiar to all rhinologists, has recently been reviewed by Alden [2] and Williams [3] and Voorhees.[4]

Specific indications for the Caldwell-Luc operation will be reflected in the case reports included in this study. According to Childrey,[5] when surgical intervention is necessary in the treatment of chronic maxillary sinusitis it should be as conservative as is consistent with cure of the disease. His operation, cristectomy, is similar to Sturman's and offers permanent aeration and drainage with an entirely satisfactory opening for subsequent observation. Childrey, however, recognized certain types of cases to which the transoral operation is best suited. Goodyear [6] reported a large percentage of cures in cases of chronic infection of the antrum if a large and well rounded nasoantral window is made, through which gauze saturated with a 5 per cent solution of iodoform

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1. In this report, the terms maxillary sinus and "maxillary antrum" will be used synonymously.

2. Alden, A. M.: The External Operation on the Maxillary Sinus, South. M. J. **31**:282 (March) 1938.

3. Williams, H. L.: Intranasal Operation for Chronic Maxillary Sinusitis, J. A. M. A. **105**:96 (July 13) 1935.

4. Voorhees, I. W.: Local Anesthesia in Radical Antrum Operations, J. Am. Dent. A. **17**:1836 (Oct.) 1930.

5. Childrey, J. H.: Cristectomy: Permanent Window in the Antrum for Chronic Maxillary Sinusitis, Arch. Otolaryng. **26**:488 (Oct.) 1937.

6. Goodyear, H. M.: Chronic Antrum Infection: Treatment by Intranasal Antrum Operation and Packing; Clinical and Experimental Results, Arch. Otolaryng. **20**:542 (Oct.) 1934.

is packed into the cavity and allowed to remain four to nine days. He stated, however, "I do not mean to imply that the Caldwell-Luc operation can be safely and totally replaced by this more conservative procedure." Williams,[3] in his report on 200 patients treated for chronic maxillary sinusitis by intranasal operation (Hempstead's modification of Mikulicz' technic), admitted certain indications for the Caldwell-Luc operation. He limited the cases in which the operation is indicated, however, to those in which the presence of a malignant growth is suspected, those in which sequestration has occurred and those in which sufficient tissue has not been removed by the intranasal operation. According to Fenton,[7] "If sinus operations have to be done on adults, let them be complete and thorough, removing all infected membrane. After this has been done, clean granulation tissue covers the denuded base, and within a few weeks normal membranes covered by ciliated epithelium, usually thicker and more fibrous than before, will be found lining the operated cavity." Ipolyi[8] expressed the belief that "radical operation should be performed only in those cases in which irreparable tissue change has already occurred in the mucosa." He further stated that in his experience the Caldwell-Luc technic provides the best means of avoiding anesthesia in the teeth.

When a complete study of a case has been made, an intranasal operation may be performed if it is considered adequate in itself. If not considered adequate, it is useful as a preliminary to a subsequent complete surgical procedure in the treatment of a subacute exacerbation of a chronic condition. The value of intranasal surgical intervention in the treatment of chronic infection of the maxillary sinus is unquestioned, but the present study is concerned mainly with cases of this disease in which more radical treatment was required. Nineteen patients on whom the Caldwell-Luc operation was performed had previously had an intranasal operation. The series analyzed for this report includes 70 cases of chronic maxillary sinusitis, in 15 of which the condition was bilateral; the Caldwell-Luc operation was performed in each instance, making a total of 85 operations.

LABORATORY FINDINGS

The diagnosis, chronic maxillary sinusitis, was confirmed by roentgen examinations alone in 36 cases and by roentgen examination with and without the use of iodized poppyseed oil in 34. Bacteriologic examinations were made in all cases, and in 51 the presence of strepto-

7. Fenton, R. A.: Modern Views About Sinus Infection, J Indiana M. A. **28**:12 (Jan.) 1935.

8. Ipolyi, F.: Indikationen der chirurgischen Behandlung von Kieferhöhleneiterungen, Monatschr. f. Ohrenh. **70**:401-404 (April) 1936.

cocci was revealed. In the remainder there was evidence of mixed infection. Careful nasal cytologic study was carried out in 40 cases; in 9 of these a large percentage of eosinophils was present, and in all of the group in which eosinophils were in preponderance the cultures showed some form of streptococcus. Of those cases in which a pathologic study was made of tissue removed, there was evidence of polyps or polypoid degeneration of the mucous membrane in 48, of cystic polypi in 6 and of carcinoma in 1.

DENTAL COMPLICATIONS

In cases of chronic disease of the antrum, dental complications should always be investigated. Shea [9] stated: "A maxillary sinus may be infected directly by either acute or chronic infections of the teeth immediately adjacent to the sinus or indirectly because of lowered resistance as the result of dental infections." Hall and Thomas [10] reported cases demonstrating that antritis of dental origin in an early stage responded satisfactorily to treatment by lavage, although that in an advanced stage required more radical treatment. Smith [11] has observed chronic antral infection in only a small number of persons with sound teeth. By studying the report on his anatomic and pathologic specimens, it is easy to understand why an antrum may become infected from a diseased tooth even though the root apex may not extend through the antral floor, since only a thin lamina of bone may intervene. When such a tooth is extracted, infection is likely to extend into the sinus. Hajek [12] stated that this may occur even with thick bone. Waldron [13] urged the elimination of all underlying dental infection before the undertaking of operative procedures for the relief of chronic maxillary sinusitis. This practice was adhered to in the care of the patients whose cases are reported here.

In this series there were 3 cases in which the abscess on the root of a tooth ruptured through the floor into the antral cavity. In each instance there was a perforation into the cavity when the tooth was extracted. It was found on inspection through the canine fossa at

9. Shea, J. J.: Infections of the Paranasal Sinuses of Dental Origin, Surg., Gynec. & Obst. **46**:408 (Feb., no. 2 A) 1938.

10. Hall, S. S., and Thomas, H. V.: Chronic Maxillary Sinus Disease in the Adult: Its Treatment and Prognosis, West Virginia M. J. **31**:249 (June) 1935.

11. Smith, A. E.: Empyema of the Maxillary Sinus: Conservative and Efficient Surgery, J. Am. Dent. A. **16**:1267 (July) 1929.

12. Hajek, M.: Pathology and Treatment of the Inflammatory Diseases of the Nasal Accessory Sinuses, translated and edited by J. D. Heitger and F. K. Hansel, St. Louis, C. V. Mosby Company, 1926, vol. 1, p. 265.

13. Waldron, C. W.: Maxillary Sinusitis, J. A. M. A. **85**:1530 (Nov. 14) 1925.

operation that a large area of antral lining had undergone necrosis. In 2 other cases there were granulomatous changes with degeneration of the lining membrane, although cysts had not completely developed. Two patients had polypi of dental origin, which were demonstrated with roentgenograms made after the injection of iodized poppyseed oil, and in other cases the polypi, because of the site of their attachment, might have been of dental origin. It is the opinion of some that the unavoidable trauma, such as rocking of a tooth, inflicted at the time of extraction, will cause infection to pass directly upward into the antrum, resulting eventually in chronic infection. The pathologic study in the remaining 25 cases in this series did not show definite evidence of dental origin of the infection.

SYPHILIS

Two patients who had positive Wassermann reactions were given antisyphilitic treatment for three months before operation. One of these had chronic frontal sinusitis on the same side with cortical perforation of the anterior wall of the frontal sinus. This sinus was irrigated during the period of treatment and then operated on according to the Lynch technic. The Caldwell-Luc operation was deferred until convalescence from the frontal operation was complete. In the case of the other patient operative and pathologic findings showed no unusual condition. In the cases of both, the antisyphilitic treatments were continued and uneventful recoveries recorded.

PRECAUTIONS IN SURGICAL TREATMENT

Most operators prefer local anesthesia for the Caldwell-Luc operation, since it affords better working conditions for the surgeon and lessens the possibility of pulmonary complications for the patient. Preoperative administration of pentobarbital sodium, morphine sulfate and atropine, in doses according to the individual requirement, helps considerably to insure the cooperation of the patient.

Ersner, Pressman and Ersner [14] in 1931 described an improved technic for the administration of local anesthetics, which in my experience has left little to be desired. Using the technic which they outlined, I have induced adequate anesthesia without complaint from my patients. The method includes injections at the following sites: (1) the infraorbital nerve at its foramen, (2) the posterior superior alveolar branch of the maxillary nerve, (3) the anterior incisive canal, (4) the posterior palatine foramen and (5) the middle meatus. After the

14. Ersner, M. S.; Pressman, J. J., and Ersner, W.: Local Anesthesia for the Radical Maxillary Operation: An Improved Technique of Administration, Arch. Otolaryng. **13**:379 (March) 1931.

injections a pack of gauze saturated with a 10 per cent solution of cocaine is placed under the inferior turbinate. In the injection of the posterior superior alveolar branch of the maxillary nerve, care must be exercised to avoid piercing a blood vessel with the needle point. Alden [2] reported that he blocked the second division of the trigeminal nerve by Blair's method, using a Barnhill needle. Schuster and Decker [15] have simplified the technic by using a single injection, ¼ to ½ inch (0.64 to 1.27 cm.) in depth, into the infraorbital foramen.

In an effort to avoid complications from the severing of branches of the infraorbital nerve, extensive research has been done regarding the incision. Mollison [16] stated that he used a horizontal and a vertical incision. Love [17] asserted that he favored a vertical incision, and McKenzie [18] and also Monson [19] recommended a right angle incision in order to shorten the horizontal division of the parts along the gingival line when the canine fossa is exposed. Alden [2] mentioned that he used a similar combination of vertical and horizontal incisions, making a triangular flap to uncover the site to be entered. Shapiro, Fabricant and Stephan [20] studied a series of cases to evaluate the vertical incision in relation to sensory disturbances. They recorded data on 17 patients with bilateral antral disease which necessitated operation by the transoral route. The usual horizontal incision was made on the right side, and the vertical incision was used on the left. Examination and testing of 10 of these patients one year later proved that the vertical incision offered no advantages in avoiding disturbances of sensation.

The horizontal incision was used in all the operations of the present series. Several patients had devitalization of teeth, but only 1 suffered a permanent disturbance of sensation. The patient's lateral incisor and his canine tooth on the involved side have remained anesthetized since the operation. Galvanic therapy has not improved the uncomfortable dead feeling in this area. Knight,[21] chief of the anatomy department

15. Schuster, B. H., and Decker, W. C.: Local Anesthesia in Radical Operations on the Maxillary Sinus, Arch. Otolaryng. **20**:67 (July) 1934.

16. Mollison, W. M., and others: Discussion on the Treatment of the Suppurating Maxillary Antrum, Proc. Roy. Soc. Med. **25**:1057 (May) 1932.

17. Love, A. A.: Instruments for Radical Operations on the Sinuses, Arch. Otolaryng. **19**:389 (March) 1934.

18. McKenzie, W. R.: Chronic Sinusitis: A Complete Operation, Technique and Results in Two Hundred Consecutive Cases, South. M. J. **30**:1077 (Nov.) 1937.

19. Monson, L. P.: The Right Angle Incision in the Radical (Caldwell-Luc) Antrum Operation, Arch. Otolaryng. **22**:254 (Aug.) 1935.

20. Shapiro, L. L.; Fabricant, N. D., and Stephan, R. M.: Sensory Disturbances Following Radical Operations on the Antrums, Arch. Otolaryng. **19**:303 (March) 1934.

21. Knight, H. O.: Personal communication to the author, April 29, 1939.

of the University of Texas School of Medicine, stated that members of his department have been of the opinion that a regeneration of the divided anterior and middle superior alveolar nerves accounts for the restoration of feeling in the teeth involved after operations through the canine fossae. Large [22] expressed disagreement with this theory. Hajek [12] cautioned against the resection of the infraorbital edge of the canine opening, for if one approaches closer than 1 cm. below the orbital edge the injury to the nerve frequently results in long-continued anesthesia, which is a source of discomfort to the patient, and bruising of the nerve may cause severe neuralgic pains for a long time. Also, if vigorous curettement of the antral floor is practiced, the bony canal conducting the nerves and blood vessels may be injured and the premolar and molar teeth devitalized as a result. As Buzoianu [23] has pointed out, "An untimely curettage of untransformed mucosa or of osseous walls which are unattacked by osteitis may cause secondary infection of the bone."

The removal of all diseased tissue, leaving apparently healthy tissue intact, is the surgical ideal. When such authorities as Lillie,[24] Wilkinson,[25] Mollison,[16] Russell,[26] Buckley [27] and McKenzie [18] are not all in accord on this subject, it seems advisable for one to make pathologic studies in a series of cases to determine the logical course to follow. In 10 consecutive cases, I made a biopsy of a questionable area of lining in order to compare the tissue with that of an evidently diseased area; in all instances but 1 this comparison showed a lining apparently diseased beyond repair. For a time it was my practice to remove the lining en masse in all cases, but after making a more careful study I remove only diseased and questionable lining.

Love [17] reported that he made an opening in the fossa with a small mastoid curet, being careful not to break the mucosal lining, then carefully loosened the lining around the periphery and removed it en masse if it was not too completely degenerated or too friable. Buckley [27] warned that if the antral wall is taken down too far injury to the

22. Large, S. H.: Empyema of the Antrum of Highmore, Ohio M. J. **26**:219 (March) 1930.

23. Buzoianu, G.: Denker Method in Radical Therapy of Chronic Antral Suppuration, Spitalul. **55**:227 (June) 1935.

24. Lillie, H. I.: Chronic Suppurative Sinusitis: A Point of View as to Treatment, Arch. Otolaryng. **21**:272 (March) 1935.

25. Wilkinson, A. P.: Chronic Maxillary Sinusitis: A Study of Three Hundred and Thirty-Two Operated Cases, J. Michigan M. Soc. **29**:305 (April) 1930.

26. Russell, B.: The Operative Treatment of Chronic Sinus Infection, J. Laryng. & Otol. **53**:201 (March) 1938.

27. Buckley, R. E.: How to Obviate Failures in the Results of Paranasal Sinus Surgery, Laryngoscope **44**:853 (Nov.) 1934.

superior dental artery and nerve may occur, resulting in hemorrhage or anesthesia. Mollison [16] said that he made a triangular opening, base up, to protect the nerves. A gauze-covered curet is the best instrument for removing the diseased lining, because it lessens the bleeding. It is almost impossible to strip up the normal mucous membrane with a gauze-covered curet, and any necrosis is bared for careful attention rather than scraped blindly before detection. Hajek [12] stated that injury to nerves and blood vessels is also less likely with the gauze-covered instrument.

The selection of patients who should have surgical treatment of the sphenoid and ethmoid sinuses at the time of the Caldwell-Luc operation and those whose sinuses should be left to heal spontaneously presents a problem which taxes the ingenuity of the operator fully as much as the decision regarding the removal of the antral lining. Pietrantoni,[28] in his anatomic, clinical and surgical study, made this logical statement: "The radical operation, if it is to be rational, must be completed with a systematic exploration of the ethmoid region." He found that chronic posterior ethmoiditis might be masked and that slight pressure on the instrument is sufficient for the exposure of the ethmoid cells.

The transoral is the most accessible route to the ethmoid and sphenoid sinuses and to the infraorbital ethmoid cells. It spares the middle turbinate bone, which is so essential to the physiology of the nose, according to Robison.[29] However, the operation is made more thorough by combining it with the intranasal work in these regions. The middle and inferior turbinates, however, must be spared. When Jansen proposed the use of this transoral route to the ethmoid and sphenoid sinuses, objections were raised because of the hazard to the optic nerve. Pietrantoni [28] stated the belief that this danger may be avoided by directing the instrument toward the nasal cavity, never toward the orbital wall. Russell [26] advocated locating the sphenoid ostium intranasally as a safety measure in avoiding the optic nerve. He expressed a preference for Denker's operation when transoral surgical procedure is contemplated. Sewall,[30] Russell [26] and Robison [29] have described similar approaches to the ethmoid and sphenoid sinuses, and have made suggestions for improving the exenteration of these cavities. In 12 cases of this series it was decided, after exploration

28. Pietrantoni, L.: Le sinusiti chroniche etmoido-mascellari. Rilievi anatomici, clinici e chirurgici, Oto-rino-laring. ital. **5**:457 (Sept.) 1935.

29. Robison, J. M.: Nasal Physiology and Its Relation to Sinus Surgery, Texas State J. Med. **34**:434 (Oct.) 1938.

30. Sewall, E. C.: General Surgical Principles Employed in the Treatment of Chronic Disease of the Nasal Sinuses, Ann. Otol., Rhin. & Laryng, **38**:963 (Dec.) 1929.

of a small portion of the anterior and posterior ethmoid cavities, to permit the ethmoid and sphenoid sinuses to heal spontaneously. This plan was justified in all but 2 cases, in which intranasal treatment was subsequently given. When the frontal sinus is also involved, a condition which constitutes a pansinusitis, the Lynch [31] method deals first with the maxillary sinus. After it has healed, the frontal sinus is operated on, and surgical treatment of the ethmoid and sphenoid sinuses through the frontal opening is included. In 1 of my cases, this order was reversed because of an annoying cortical perforation of the frontal sinus. In the other, the Caldwell-Luc operation was performed first.

In making the nasoantral opening in the inferior meatus, one should spare the posterior one third of the lateral nasal wall. The inferior turbinate is retracted upward, and through the nasoantral opening a large rubber tube is inserted and allowed to remain for four days if well tolerated. Iodoform gauze, ¼ inch (0.64 cm.) in width, saturated with petrolatum, is packed into any area of suspected bleeding, and the end of the gauze is threaded through the rubber tube. Through this tube, as Wherry [32] suggested, liquid petrolatum containing phenol in 0.5 per cent concentration may be instilled to increase the postoperative healing and comfort. The tube also provides an ideal route for dropping hydrogen peroxide into the cavity to soften the packing before its removal. The tube in the nasoantral opening is the best means of insuring a permanent opening, as it causes granulation to form around the periphery, to which the lining, when regenerated, adheres, leaving a smooth surface over its borders.

The incision is always closed with double-armed chromic quadruple zero catgut. The sutures are left to come out of their own accord. The edges are evenly approximated and are turned out with small mouse tooth forceps after suturing. I have used this suture successfully in cases of fistula from operation after plain catgut, silk and linen sutures had sloughed out.

POSTOPERATIVE CARE

The patient is given the sedatives necessary to insure comfort, an ice bag is applied on the side operated on and 15 grains (0.97 Gm.) of calcium gluconate is administered intravenously. Since the advent of sulfanilamide, 10 grains (0.65 Gm.) has been given every six hours for two days, to lessen postoperative infection. Autoclaved petrolatum containing phenol in 0.5 per cent cencentration is dropped through the

31. Lynch, R. C.: The Technique of a Radical Frontal Sinus Operation Which Has Given Me the Best Results, Tr. Am. Laryng., Rhin. & Otol. Soc. **26**:60, 1920.

32. Wherry, W. P.: Immediate Postoperative Technic Incident to Caldwell-Luc Operation, Ann. Otol., Rhin. & Laryng. **41**:460 (June) 1932.

tube three times a day. No other local postoperative treatment is given, except a possible irrigation on the sixth or seventh day, if indicated.

RÉSUMÉ OF CASES

The essential findings in the present series may be reported briefly. Thirty-eight of the patients were males, and 32 were females, their ages ranging from 16 to 72 years. The physical condition of the 70 patients was as follows: 19, poor; 15, fair, and 36, good. Fifty-three complained of the usual chronic sinal symptoms, which in 9 cases were complicated by allergy (asthma in 5 and hay fever in 4). Twelve of the 53 patients had bilateral sinal symptoms. Thirty of the 53 gave a history of influenza or severe head colds. Thirty-two patients had dental involvement or had had previous extractions on the involved side. In 7 patients the sinusitis resulted directly from dental infection. Four patients had symptoms identical with those of tic douloureux. A positive diagnosis of cysts or polypi was made in the case of each of these, all of whom were entirely relieved of their symptoms within three months after operation. One patient, however, had a recurrence of symptoms two years later and was referred to a neurosurgeon. In 9 cases of the so-called "silent" type, the patients were unaware of the presence of the foci of infection. In 3 of this group the infection was bilateral. The sphenoid and ethmoid sinuses were involved in 20 cases, the frontal sinuses in 2. Three patients had foreign bodies in the antrum: (1) a piece of shrapnel, (2) the tip of a Thornwald drill and (3) the root of a second molar tooth, which had been forced through the antral floor and the presence of which was attributed to surgical accident rather than to dental origin. The duration of the symptoms, except in the cases in which a foreign body was present, varied from two months to twenty years.

RESULTS

A tabulation of the results in the present series has been made possible by continued observation of the patients, most of whom have been followed. Fifty-eight were cured, 7 showed improvement, and 2 were not benefited. Of the 58, 1 died from cancer a year after operation. One died from cardiac disease two years later. Three of the patients classified as cured required second operations through the canine fossa, and 2 had operation on the sphenoid and ethmoid sinuses on the side of the maxillary involvement six weeks after the Caldwell-Luc operation was performed. At the time of the first operation, in each case, it was thought that the infection in the sphenoid and ethmoid sinuses would heal spontaneously. Three patients whose condition was considered satisfactory at the time of discharge were lost to follow-up. In each of the 9 cases of maxillary sinusitis of the so-called "silent type"

previously mentioned, the sinusitis was considered to be the focus of infection. After operation, 6 of the patients showed sufficient symptomatic improvement to justify the operation.

Of the 9 patients who had antral disease coexisting with allergy, 2 with asthma obtained gradually increasing relief as the antrum healed and have remained well for five and eight years. Another patient with asthma and hay fever experienced a reduction in the frequency and duration of the asthmatic and hay fever attacks and has shown 75 per cent improvement during the nine years following the operative procedure. Two others showed temporary improvement. The other 4 had no change in their allergic status.

The condition of the antral cavity after a surgical procedure is not always all that could be desired. It is better, however, after a Caldwell-Luc operation that was indicated than if an inadequate surgical operation had been performed. If it is practical to leave mucosa around the ostium, the physiologic function is better, as King [33] observed from Hilding's experiments with india ink on a patient who had a large nasoantral opening; the india ink flowed past the artificial window and out through the normal opening. If the lining is regenerated, according to Knowlton and McGregor,[34] Boling [35] and Wright,[36] this should also be true in regard to the radically treated patients. Grove [37] noted the incompatibility of physiologic sinal function with chilling, ventilation, chemical irritation and metabolic fluctuation, any of which may be responsible for a break in the defense mechanism of the mucosa. He was not speaking of postoperative conditions but his theory should be applicable to the condition of a patient who has been operated on, after regeneration has taken place.

SUMMARY

The intranasal procedures are useful and preferable if ample to meet the therapeutic requirements of the existing disease. However, in the cases reported the conditions were considered to be beyond the scope of intranasal surgical treatment.

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33. King, E.: Clinical Study of the Functioning of the Maxillary Sinus Mucosa, Ann. Otol., Rhin. & Laryng. **44**:480 (June) 1935.

34. Knowlton, C. D., and McGregor, G. W.: How and When the Mucous Membrane of the Maxillary Sinus Regenerates, Arch. Otolaryng. **8**:647 (Dec.) 1928.

35. Boling, L. R.: Regeneration of Nasal Mucosa, Arch. Otolaryng. **22**:689 (Dec.) 1935.

36. Wright, C. F.: Radical Maxillary Sinus Operation, Caldwell-Luc: Summary of Clinical and Histological Observations on One Hundred Patients, Arch. Otolaryng. **12**:63 (July) 1930.

37. Grove, W. E.: Conservative Treatment of the Nose and Accessory Sinuses, Ann. Otol., Rhin. & Laryng. **43**:988 (Dec.) 1934.

Syphilis, dental infection and the general condition of the patient should be studied and given attention before operation.

When allergy is present with chronic maxillary disease, the patient should receive the same surgical attention as a nonallergic person.

The literature is replete with surgical precautions. I was prompted to review and record them after one of my patients suffered a permanent disturbance of sensation in two teeth after operation. In this connection, the horizontal incision is no more hazardous than the vertical.

All patients with tic douloureux should have an exhaustive study of the maxillary antrum on the involved side before surgical procedure on the fifth nerve is resorted to.

Histopathologic examination of all tissue removed should confirm or contradict the justification for its removal by the Caldwell-Luc operation. By making such examinations and correlating the pathologic findings with the diagnostic data, otolaryngologists can improve their judgment in the selection of patients who should be operated on by the transoral method.

1300 Eighth Street.

THE LARYNX IN THE TUBERCULOUS PATIENT

ROBERT M. LUKENS, M.D.
PHILADELPHIA

The material in this paper is derived from the study of tuberculous patients in the tuberculosis dispensaries of the Jefferson Medical College Hospital and the Henry Phipps Institute and in private practice. Bed patients and those in whom the disease is in an advanced stage are considered only occasionally, as the purpose of this paper is a discussion chiefly of the laryngeal conditions in ambulatory patients in whom the disease is in an early stage.

In this era of preventive medicine, it is surprising that general practitioners pay so little attention to the larynx of the patient with pulmonary tuberculosis in the early stage. Some of them retain the old idea that once laryngeal involvement takes place the patient is doomed, and for that reason the laryngologist frequently is not consulted until symptoms of advanced disease occur. It is my experience in private practice that very few patients with tuberculosis are referred to the laryngologist for prophylactic laryngeal care.

In the dispensaries of institutions for the care of patients with tuberculosis, where routine examinations of the nose and throat are made, the patient can have the benefit of laryngeal prophylaxis. It is in the dispensaries that laryngologists are given the opportunity to see and treat tuberculous laryngitis in the early stages and in many cases to prevent its occurrence.

Many patients with advanced pulmonary and laryngeal tuberculosis are seen in the dispensaries, but most of these are sent to sanatoriums as soon as possible. The greatest hope for the ultimate reduction of laryngeal complications of tuberculosis rests in the prophylactic and active treatment of patients seen at dispensaries. The dispensary has been aptly called the preventorium.

Every case of pulmonary tuberculosis is a potential case of laryngeal tuberculosis. There is no particular stage of pulmonary tuberculosis at which laryngeal involvement makes its first appearance; consequently, one should be on the lookout for it not only in the advanced stages of the pulmonary disease but at fairly regular intervals from the beginning throughout the duration of the disease, regardless of the absence of laryngeal symptoms. In other words, a patient with pulmonary tuberculosis should be under the joint care of a physician and a laryngologist.

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There is nothing to foretell cancer of the larynx. The first symptom must make its appearance before laryngeal disease is suspected. However, tuberculosis of the larynx, except in a few instances, is heralded by pulmonary disease. Occasionally there are cases in which the laryngeal symptoms precede the pulmonary symptoms.

In established pulmonary tuberculosis, the larynx is rarely normal. In most cases the larynx shows inflammation in various stages, particularly if cough is a prominent symptom.

While nasal infections and sinusitis probably are no more common in tuberculous than in nontuberculous persons, they have a greater significance in a tuberculous patient because of the lowering of the resistance of the larynx.

Chronic catarrhal laryngitis probably is the most common laryngeal lesion seen in tuberculous patients in the dispensaries and is frequently associated with chronic nasal infections and chronic pharyngitis.

Laboratory experiments have shown that tubercle bacilli can enter the submucosa through intact epithelium, but I cannot help feeling that it is more than likely that the bacilli enter the submucosa of the larynx at the point where denudation of the epithelium has occurred as a result of inflammatory desquamation, the epithelium subsequently regenerating over the invaded area.

The tubercle is nature's defense against the invading bacilli, and if general and local resistance are maintained the process is stopped, but if local resistance is broken down by trauma or infection the lesion may progress until it can be recognized clinically.

Healthy mucosa is naturally resistant to the tubercle bacillus, but in the presence of local inflammation and infection its resistance may be broken down. When nasal and pharyngeal infections remain untreated, they are a constant source of irritation to the larynx, predisposing the laryngeal tissues to an initial invasion, and after the development of definite tuberculous lesions healing of the laryngeal tissues may be retarded or stopped entirely.

Such conditions as chronic hypertrophic rhinitis, pronounced deviation of the septum or the presence of nasal spurs or of nasal polypi interfere with nasal respiration and ventilation and produce mouth breathing. Infections of the nose and sinuses, frequently a result of the conditions named, throw an added burden on the laryngeal mucosa and in the presence of existing tuberculous lesions may result in secondary infections of the ulcerations.

Tuberculous ulcers tend to heal, but when secondarily infected by other organisms they are likely to spread, and healing is delayed if not actually stopped.

This I feel is one of the reasons why in some cases laryngeal tuberculosis progresses in spite of the fact that the pulmonary tuberculosis

has been arrested. In my experience, patients with laryngeal tuberculosis in an early stage and even those with ulcerative lesions do well when the nasal and pharyngeal infections are taken care of and mild oil preparations are instilled into the larynx and trachea.

SYMPTOMS

The apparent absence of symptoms in many cases eventually impresses the laryngologist who does much work on patients with tuberculosis. Infiltrations and ulcerations on the posterior surface of the epiglottis, which are more common than generally supposed, on the posterior wall, at the angle formed by the junction of the vocal cords with the arytenoid, on the inner surface of the arytenoids and the ventricular bands and occasionally on the vocal cords may produce no symptoms at all.

Patients are frequently unaware that their voice has altered, so that a history of mild hoarseness or roughening of the voice is not obtained. One of my patients when asked how long he had been hoarse declared that he was not hoarse and that his voice "had always been that way." On examination, a granuloma, 2 mm. in diameter, was found at the anterior commissure. After removal of the granuloma by direct laryngoscopic procedure, he was surprised by the clearness of his voice. The hoarseness had developed so gradually that neither he nor his associates had noticed the change. Hoarseness and cough are common symptoms in so many respiratory infections that their value is diminished as laryngeal symptoms of undiagnosed tuberculosis in regions where "colds" are prevalent. Even when hoarseness and cough are prolonged over the usual time for nontuberculous infections of the respiratory tract, tuberculosis is too frequently overlooked. Hoarseness when its development is gradual is often unnoticed by the patient, and frequently the vocal alteration is first noticed by the laryngologist during a routine examination of the nose and throat. In these days of universal cigaret smoking, chronic cough is frequently thought to be a "cigaret cough," and until more serious symptoms arise, little attention is paid to it by the average patient with undiagnosed tuberculosis.

Sensations of dryness and irritability of the throat are frequent in those patients having nasal and pharyngeal inflammations and seem to be present when early tuberculosis is associated with chronic laryngeal inflammation.

Vocal weakness is present mainly in asthenic patients who have advanced pulmonary lesions.

In the tuberculous patient, dysphagia and odynphagia usually are late symptoms, although an occasional patient has these symptoms before consulting a laryngologist.

EXAMINATION

There is only one way in which predisposing conditions can be evaluated and an early diagnosis of tuberculosis of the larynx can be made, and that is by careful and frequent examinations of the larynx, beginning as soon as the pulmonary disease is diagnosed.

After all that has been written in the past fifteen years on the early recognition of tuberculosis of the larynx it is surprising how often this disease in an advanced stage is seen for the first time in the course of routine examination of the throats of patients with pulmonary tuberculosis.

Frequently these patients have consulted many physicians who failed to examine the larynx or who felt that the laryngeal symptoms were not sufficiently severe to indicate serious laryngeal disease.

One examination is not sufficient to rule out laryngeal involvement in tuberculous patients. Small early tuberculous lesions may be obscured by simple chronic laryngitis, and it is only by repeated examination that the obscured tuberculous lesion can be discovered in an early stage.

If the laryngologist can see only the posterior half of a larynx and finds that portion free of lesions, he should not feel satisfied that the larynx is free from tuberculosis by reason of the fact that tuberculosis most commonly attacks that portion of the structure. The following case illustrates this point.

A young man had been under private treatment for a considerable time for pulmonary tuberculosis. During the course of his pulmonary disease, persistent hoarseness developed. He was referred to a laryngologist for examination of his throat and for treatment. According to the patient, his condition was diagnosed as chronic laryngitis and his throat was treated with sprays. I saw him routinely at a hospital dispensary. His throat was very sensitive, and it was impossible to see the anterior half of his larynx because of drooping of the epiglottis. The part visible to the laryngeal mirror showed chronic laryngitis with no evidence of tuberculosis. However, after the throat was anesthetized and the epiglottis retracted, a small granuloma was revealed, attached to the margin of the anterior third of the right vocal cord. The laboratory report was "tuberculoma." There was no recurrence.

It is necessary to see not only the full length of the vocal cords but also the posterior surface of the epiglottis, as early individual tuberculous lesions have occurred in this region of the larynx.

The larynx of a tuberculous patient may be subject to any of the lesions found in the larynx of a nontuberculous patient. These will not be discussed, as they are entirely independent of tuberculosis except as sources of irritation to the larynx and must be dealt with as in nontuberculous patients. The differential diagnosis between hypertrophy of the mucosa of the posterior wall and enlargements of the arytenoids and tuberculosis of these structures sometimes presents difficulties.

Many patients have thickening of the mucosa of the posterior wall and bilateral swelling of the arytenoids due to chronic inflammation, but these are not to be confused with tuberculosis. In my cases they were usually associated with nasal and pharyngeal infections. These seemed to be almost permanent changes and usually persisted for a long period. Tuberculous infiltration of the posterior wall and arytenoids either improved or became worse in the course of time, and in the arytenoids the changes were not symmetric as a rule. Frequently when little more than redness of the vocal process was visible, the subglottic portion of the cord showed definite tuberculous infiltration.

TREATMENT

Laryngologic treatment of a tuberculous patient comprises treatment not only of a tuberculous lesion of the larynx but also of all existing diseases of the nose and throat. Infections of the nose can safely be treated even in advanced pulmonary tuberculosis, but nasal conditions requiring surgical intervention may be risky. However, if such conditions of the nose are diagnosed during the early stages of pulmonary tuberculosis or during quiescent periods, there is little risk attached; therefore, these conditions should be attacked at the earliest opportunity. Examination of the nose, throat and larynx when pulmonary tuberculosis is first diagnosed usually provides time to do necessary operations on the nose and throat before operations are contraindicated by advanced pulmonary lesions.

During this period diseased tonsils may be removed with the region under local anesthesia, although some authorities believe that operation on the tonsils is contraindicated in any patient who has the least pulmonary activity. Diseased tonsils are a distinct menace when the larynx is tuberculous, particularly if they are much hypertrophied and cryptic. When the laryngeal lesion is advanced, operation on the tonsils is dangerous. In my experience, electrocoagulation when done slowly and on a small portion of tissue at a time has never had a bad effect on either the laryngeal or the pulmonary lesion, but on the contrary both the lung and the larynx have shown improvement. However, indications for surgical intervention should be considered individually in each case, and operation should be done only when approved by both the internist and the laryngologist.

In the past, tracheotomy has been advocated by a few writers as a means of enforcing vocal rest, but in the few cases in which I have had to do emergency tracheotomy, the wound has always become infected and the secretions from the lung messy. In only 2 cases did the larynx show improvement. The 2 patients in whom the larynx healed were tracheotomized because of threatened asphyxia due to sudden bilateral paralysis. The lesions were only moderately advanced and involved only

the vocal cords and the posterior wall. Complete vocal rest was obtained through the paralysis of the vocal cords, and the pulmonary secretions were detoured through the tracheotomy tube. Although both patients died from the pulmonary disease six months later, the laryngeal lesions healed without other local treatment.

Artificial Pneumothorax.—Patients under treatment with artificial pneumothorax seemed to do well. By lessening cough and expectoration, artificial pneumothorax relieves the larynx of irritation and contamination by the pulmonary secretions.

Electrocauterization.—The electrocautery is the accepted means of local treatment for laryngeal tuberculosis and is one of the most effective means available, yet I feel it is a mistake to advise its use in all cases of laryngeal tuberculosis. It is comparatively easy for a laryngologist experienced in the treatment of laryngeal lesions to apply the cautery, but a novice who has had little experience in the treatment of tuberculosis can do a great deal of harm with the apparatus. I have observed several cases in which the cautery was used on unsuitable larynxes. One of these patients required emergency tracheotomy; others were made worse by edema of the larynx and dysphagia. Patients to be treated with the electrocautery should be selected carefully and as small an amount of tissue as possible destroyed at one sitting.

Other Therapeutic Measures.—There should be no great haste in treating even advanced laryngeal tuberculosis. In the average case this is a slow chronic disease, and if it is treated with due regard to the condition of the larynx and the patient in general a great deal may be accomplished. Hasty and too vigorous local treatment often defeats its own purpose by producing unnecessary trauma and overburdening a larynx which already is barely holding its own.

Since the old days when it was considered necessary to treat tuberculous lesions of the larynx with vigorous applications of lactic acid and solution of formaldehyde, I have been conservative in my treatment of this disease. My routine has been to treat nasal and pharyngeal infections and to allay laryngeal and tracheal irritation.

Much of the cough in pulmonary tuberculosis is unnecessary and tends to keep up laryngeal inflammation. This can be relieved to a great extent by intratracheal instillations of monoparachlorophenol in oil or 0.5 per cent menthol in oil. Many patients with laryngeal tuberculosis in an early stage have required no other local treatment. I feel that the unnecessary cough, particularly the unproductive irritative type, does more harm to the larynx than excessive use of the voice.

I prefer direct treatment with chaulmoogra oil, full strength, for tuberculous infiltration and ulceration and electrocauterization for the reduction of turban epiglottises and piriform arytenoids in suitable cases.

Vocal rest is the one treatment that is indispensable and should be complete. During periods of hoarseness or voice fatigue, even if there is no demonstrable lesion, vocal rest is indicated. Usually these periods are of short duration. When tuberculous lesions are present, complete vocal rest is indicated. Many patients either cannot or will not practice vocal rest for a long period. In their cases I frequently compromise by permitting conversation at meal times. Apparently whispering loud enough to be heard tires the throat more than low-pitched talking.

SUMMARY

Laryngeal examination of the tuberculous patient is too often postponed until laryngeal symptoms make their appearance. If one hopes to reduce laryngeal complication to a minimum, regular routine examinations of the larynx should be made as soon as pulmonary tuberculosis is diagnosed.

In the majority of patients seen at a dispensary, laryngeal symptoms are slight or absent except in those in whom pulmonary tuberculosis is advanced or care has been neglected.

The predominating laryngeal lesion is chronic laryngitis, frequently associated with nasal infection and pharyngeal inflammation.

Surgical treatment of the nose and throat to correct conditions interfering with normal nasal breathing and producing nasal infections which may predispose the larynx to tuberculosis can be done if laryngologic examinations are made very early in the course of pulmonary disease.

In the great majority of cases in which removal of tonsils is required, tonsillectomy by cutting is contraindicated because of the advanced stage of the pulmonary disease or the presence of tuberculosis of the larynx. In some of these cases electrocoagulation of the tonsils, carefully performed, helps to improve the general condition of the patient and does not have a bad effect on the laryngeal or pulmonary lesions.

Local treatment to the larynx should not be too vigorous, but the larynx should be given every opportunity to recover from tuberculous lesions by the elimination of sources of secondary infection, by the reduction of chronic laryngeal inflammation and by the elimination of unnecessary, unproductive cough.

Tuberculous infiltrations and ulcerations when seen early in the course of the disease have a tendency to heal if the patient's general condition is good, the pulmonary disease is under control and the nose and throat are kept in a healthy state.

Specific local treatment for individual lesions should be determined by the condition of the larynx in each case.

1923 Spruce Street.

PRACTICAL POINTS IN THE TREATMENT OF SPHENOIDITIS

FRED W. DIXON, M.D.
CLEVELAND

Proper treatment of any pathologic condition presupposes a correct knowledge of the anatomy of the region involved as well as of the structures surrounding that region. The sphenoid sinus, on account of its inaccessibility, is frequently overlooked and rarely treated. All the posterior sinuses are favored by their dependent drainage and the position of their natural ostiums. That they are invaded, however, is shown by the investigations of Teed.[1] In summarizing his report of 1,060 clinical cases of sinusitis, he stated that the sphenoid sinus was involved in only 15.7 per cent. When, however, the autopsy reports in 1,184 cases were analyzed, it was found that the sphenoid sinus had been diseased in 250, or 22 per cent of these cases.

This, the most variable of all the sinuses in shape, size and drainage, may be in relationship to all three of the cranial fossae. Its walls may come into close contact with the optic, the oculomotor, the trochlear, the maxillary division of the fifth and with the vidian nerves. Usually the cavity is separated from the internal carotid and the sphenopalatine arteries, as well as from the cavernous sinus, by only a very thin partition of bone. Its capacity may vary from ½ minim (0.05 cc.) to ½ ounce (14 cc.). The sinuses may be almost equal in size, or one may be infantile, while its well developed mate is enormous and indented by all the previously mentioned important structures on both sides of the skull. This variable structure when diseased may thus, by its very location, produce a variety of symptoms. The thickness of the wall depends on the development of the sinus. The inferior wall, or floor, is usually of greater density, while the remaining walls may be of tissue paper thickness, 0.5 mm. being the average measurement of the fully developed bony wall. The anterior wall may slope down and outward, or may slope up and posteriorly. I shall later show that on this fact may depend the success of catheterization.

In an examination of 3,200 macerated sphenoid sinuses, it was noted that 7 per cent showed a large cylindric ridge covering the optic nerve

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1. Teed, T. W.: Meningitis from the Sphenoid Sinus, Arch. Otolaryng. **28**: 589 (Oct.) 1938.

on the superolateral wall.[2] On the posterosuperior surface, the sella turcica may be covered by bone of parchment paper thickness.

Dehiscences in the bone have been observed by various investigators. While such gaps may be common in the bony walls, in an examination of 300 fresh specimens I have not found them constant in the membranous covering. Since both vein and artery pass through the bone to the cranial cavity, I doubt if these dehiscences play an important part in infections of the brain. The dura is resistant and unless disturbed usually escapes involvement. In Teed's 28,400 cases in which autopsy was reported, it was found, in comparing the incidence of intracranial complications from rhinogenous and otogenous complications, that there were 7 instances of rhinogenous and 109 of otogenous complication, or that intracranial complications from the ear were fifteen times as frequent as those from the nose. However, it is extremely hazardous to curet the sphenoid cavity; there is a possibility, first, of injuring this important structure, or, second, of spreading the infection. The interior of the sphenoid cavity may be divided into separate cells. These may have their separate ostiums, draining independently, or they may drain into each other. Bony and membranous septums were found in 32 per cent of the sinuses examined. These divided the cavity into every possible shape. The most common septum joined the superior and the inferior wall posteriorly and, as such, would not greatly hinder either operation or drainage. However, if surgical procedure on the sinus is necessary, they should be considered. Barnhill expressed the belief that these interfere with drainage and that it is frequently impossible to reach and relieve the sphenoid sinus by surgical intervention. Schaeffer stated that these recesses may retain infectious material despite the establishment of good drainage surgically and warned of the danger of spreading infection by curetting these cavities. While the sphenoid sinus is usually in close contact with the optic nerve, a posterior ethmoid cell may, at the same time, partially surround it, so that neuritis of the optic nerve may be caused by infection in either or both of these cavities.

On account of the location of the sphenoid sinus, it is often difficult and in some cases impossible to obtain a roentgenogram of it. If there is doubt, it is advisable to attempt to catheterize the cavity and slowly instill warm iodized oil. A clue as to the size of the cavity may be obtained by having the patient note the overflowing of the oil. This procedure can usually be accomplished and will indicate the treatment necessary.

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2. Dixon, F. W.: A Comparative Study of the Sphenoid Sinus, Ann. Otol., Rhin. & Laryng. **46**:687 (Sept.) 1937.

In a study of 300 fresh skulls I was able to catheterize or puncture 75 per cent of the sphenoid sinuses.[3] In 7 per cent of those in which this procedure was successfully carried out the cavity was so shallow or the ostium so close to the roof that it was impossible to tell that the sphenoid has been entered. This leaves 70 per cent in which the attempt was successful. Canfield [4] stated that he is able to catheterize 90 per cent of normal sphenoid openings. This probably includes puncture of the membranous ostium. It was found that the angle formed by the junction of the turbinates and the septum, rather than the size of the ethmosphenoid recess, determined successful catheterization. The average angle normally made in the 300 fresh specimens was 30 degrees. My failures were in those instances in which the angle was less than 30 degrees. No difficulty was encountered when the angle was over 30 degrees. While I do not advocate irrigation of an acutely infected sphenoid sinus, I believe it is perfectly safe when the acute phase has subsided. A knowledge of the anatomy plus persistence and proper shrinking will usually yield success. Failures have been due to one or the other of three causes: (1) a deflected septum interfered with the passage of the catheter; (2) the ethmoseptal angle was too sharp, or (3) the ostium was abnormally placed.

It is preferable to use a small thin flexible catheter with a shoulder 7 cm. from its tip. This is bent with an arch 5 to 10 mm. high. This catheter may be used for a probe and the slope of the anterior wall noted. The range of search of the ethmosphenoid recess is also increased over that which is possible with a catheter bent at an angle similar to the bend of a eustachian tube–shaped catheter. In many cases a thin membranous wall will probably be punctured. It is not advisable to use force unless the roentgen picture shows a thin-walled cavity. If an operation is deemed advisable, the operator should bear in mind the close proximity of the previously mentioned important anatomic structures, the dangers of curetting the cavity and the danger of hemorrhage from injury to the sphenopalatine artery found on the under surface of the floor. This artery crosses from without inward and lies in the mucous membrane on the anterior-inferior surface. If the floor is to be removed, the mucous membrane should be freed from the bone and the artery ligated. If the artery is to be ligated transantrally, it should be approached at the external surface of the antrum. If the nasal septum interferes, a submucous resection should be the preliminary step. The removal of the

3. Dixon, F. W.: Catheterizing the Sphenoid, Arch. Otolaryng. **30**:994 (Dec.) 1939.

4. Canfield, N.: Clinical Recognition and Treatment of Chronic Sphenoiditis, Kentucky M. J. **39**:284 (July) 1938.

middle turbinate or of the posterior half of the middle turbinate follows. This approach is, of necessity, by way of the posterior ethmoid cells, which may be the source of the infection.

If a permanent window is desired, it is safe to remove the anterior sphenoid wall. If the mucosa is freed on both surfaces and the bone removed, there will be less tendency for the window to close. There is to be considered, however, the effect of a large window on the delicate cilia. It has been estimated that the air in the sphenoid sinus changes once in an hour. With the anterior wall removed, the cilia will probably be affected by every blast of inspired air. Contraindications of surgical treatment to the sphenoid sinus are similar to those governing treatment of other paranasal sinuses.

Whether the sphenoid sinus is approached by the external or the internal route will depend on the skill of the operator. A carefully performed, well done intranasal operation will expose the cavity as well as the external route with less deformity. No operation, either external or internal, will completely expose the sphenoid cavity in all cases.

DOES HEAT CREATED BY THE DULL DENTAL BURR DURING LABYRINTHINE FENESTRATION INHIBIT OSTEOGENESIS?

MATTHEW S. ERSNER, M.D.
AND
DAVID MYERS, M.D.
PHILADELPHIA

During the past two and a half years the surgical treatment of deafness has attracted worldwide attention. Monumental work has been recorded by Holmgren, of Sweden, Sourdille, of Paris, and Lempert, of this country.

In recent months the operation for otosclerosis has been so frequently described that another description of the procedure would be pointless.

The principle of the operation is fenestration of the labyrinth. The despair in connection with it is the closure of the fenestra.

The technical problem of fenestration of the external semicircular canal is practically solved so far as the fenestra is concerned. It is irrelevant whether the scraping method of Holmgren is employed or whether the Lempert method using the electric dental drill with a dull polishing dental burr is followed. It is not the mechanics of the formation of a fenestra that baffles the otologist; it is the osteogenesis and the closure of the fenestra that continue to present the perplexing problem.

There is a macroscopic difference in the appearance of the bone after the fenestra is formed by the Holmgren method as compared with its appearance after the use of the Lempert method. The scraping technic of Holmgren leaves the bone dull, and the burring method of Lempert leaves it shiny.

It has been hypothesized by some that osteogenesis has been retarded, or, in fact, inhibited by the heat which was created by the polishing burr during labyrinthine fenestration.

Canfield [1] in a recent report stated that osteogenesis is inhibited by the use of the dull dental burr. However, he himself raised the following questions, which we quote verbatim: "Has the bone been affected differently by the two methods? Has the mechanical process changed the bone? Should heat produced by friction of the drill be considered?

Presented before the staff of the Department of Otology of Temple University, April 12, 1940.

1. Canfield, N.: Labyrinthine Fistulas, Arch. Otolaryng. **30**:50-62 (July) 1939.

Does the drill injure bone cells which are not removed? Has the bone's power of regeneration been diminished?"

Canfield concluded that the use of a sharp instrument encourages osseous regeneration while the use of the dull burr inhibits any reformation of bone.

Since we cannot substantiate Canfield's observation in relation to heat, and since we have found that the sharp burr creates more heat than the dull, we have felt that it would be worth while to measure the degree of heat that is actually produced by the polishing process of the dull dental burr and evaluate its significance.

Accordingly, the following experiments were performed by us, with the technical assistance of Dr. Heney. Electrocouples were employed, and the heat produced by the dental drill was measured.

REPORT OF EXPERIMENTS

EXPERIMENT 1.—The mastoid of a cadaver was excavated, and the external semicircular canal was visualized. A small hole was drilled in the external limb of the canal, immediately beneath the area to be fenestrated. A platinum electrode attached to an electrocouple was inserted into the opening.

In the cadaver, in a dry field and without circulation, which should help to dissipate the heat, the maximum degree of change was 12.6 degrees (F.) and then 8 degrees (F.).

EXPERIMENT 2.—A. The mastoid bulla of a cat (under pentobarbital sodium anesthesia) was exposed; the surface heat of the mastoid bone was measured, and this was found to be 97.5 F. The dental burr was then moved upward and backward ten times over a small area of the cortex (corresponding to the size of a fenestra), and the surface temperature, in the presence of considerable bleeding, was 99.5 F., a change of 2 degrees (F.).

B. A small dull hole was made immediately beneath the cortex, and a platinum electrode was inserted. The burr was then moved over this area a varying number of times, as in the fenestration technic.

The number of times that the burr was run over the site of the fenestra varied from one to fifteen or twenty, depending on the dryness of the operative field.

The greatest change in the temperature immediately beneath the burred surface was 3.5 degrees (F.), after the drill was applied fifteen times.

EXPERIMENT 3.—The mastoid bulla was exposed in a cat (under pentobarbital sodium anesthesia). The promontory was exposed. A small platinum electrode was inserted into the oval and the round windows. The purpose of this was to measure the change in intralabyrinthine temperature which occurred as the result of the drilling on the surface.

These experiments were repeated many times with identical results.

From the measurements it can be seen that the amount of heat produced in the bone is slight. It may be conjectured that this amount would have little effect on the prevention of healing and the formation of new bone.

EXPERIMENT 4.—Cauterization and electrocoagulation at red heat above the burr hole were employed on the labyrinths of cats.

In repeating this experiment we found that we were enabled to increase the temperature of the bone significantly. The rise in temperature depended on the amount of heat, which, in turn, increased the temperature of bone from 40 to 50 degrees (F). We are inclined to believe that such a rise in temperature

TABLE 1.—*Measurement of the Temperature of the Surface of the External Semicircular Canal of a Cadaver*

Condition	Average Temperature (F.)	Average Change in Temperature, Degrees (F.)
Before starting to drill	72	
Immediately after drilling	85.5	12.6
While drilling near burr hole	80	8

TABLE 2.—*Measurement of the Temperature of the Surface of the Occipital Bone*

Condition	Average Temperature (F.)	Average Change in Temperature, Degrees (F.)
Before any drilling	94.5	
Burr applied once	95	0.5
Burr applied 4 times	95.5	1.0
Burr applied 6 times	95.5	1.0
Burr applied 10 times	96	1.5
Burr applied 15 times	98	3.5

TABLE 3.—*Measurement of the Intralabyrinthine Temperature Just Beyond the Round Window and the Oval Window*

Condition	Average Temperature (F.)	Average Change in Temperature, Degrees (F.)
Before drilling	92.5	
Burr applied to promontory 6 times	93.5	1
Burr applied to promontory 10 times	94.5	2
Burr applied to promontory 15 times	94.5	2
Saline solution applied to surface; burr used 15 times	90 to 95	3
Burr used 20 times	89.4 to 92.5	3.1

would tend to retard or inhibit osteogenesis. This observation coincides with that of the Guggenheims.[2] The terrific heat produced by electrocoagulation and cauterization would no doubt have a detrimental effect also on the membranous labyrinth.

SUMMARY OF EXPERIMENTS

On cadaver material (experiment 1) the change in temperature as a result of the use of the dull dental burr varied from 8 to 12 degrees.

2. Guggenheim, L. K., and Guggenheim, P.: Artificial Fistula in Cases of Otosclerosis, Arch. Otolaryng. **32**:1-15 (July) 1940.

Experiments 2 and 3 were repeated with identical results. The temperature at no time showed an increase of more than 3 degrees. It therefore may be conjectured that since the variations in temperature created by the dull dental burr were so insignificant, it would hardly be conceivable that the heat produced by the burr would play an important role in osteogenesis.

Experiment 4, on the other hand, did show a significant increase in the temperature of the bone. (At some future date we hope to present a supplementary report, showing the histologic changes in the membranous labyrinth produced by the extreme heat of electrocoagulation and cauterization.)

CONCLUSIONS

We regret that we cannot subscribe to the hypothesis that the heat of a burnishing burr inhibits osteogenesis. Since the heat created by the smooth, dull burr did not at any time exceed 3 degrees (F.), as was shown in our experiments, we cannot attribute to this technic the success of some of our fistula operations for otosclerosis, nor can we attribute our failures to it.

The fact remains that, although the difficulties attending the surgical treatment of otosclerosis have been overcome, the end result, improvement in the hearing of patients with otosclerosis, is only temporary in the majority of cases. And, so long as the maintenance of the fistula and the inhibition of osteogenesis remain the crux, or, shall we say, the enigma, of the problem, we are of the opinion that scientific experiments will have to be continued until permanent patency of the fistula has been achieved.

1915 Spruce Street.

PRESENT STATUS OF VITAMINS IN RELATION TO EIGHTH NERVE AND CONDUCTION DEAFNESS

GRANT SELFRIDGE, M.D.
SAN FRANCISCO

I [1] have described elsewhere the effects of the entire vitamin B complex, prepared from California rice bran, on the eighth nerve and have stated that its use on human beings was prefaced by the finding of definite evidence of nerve degeneration in rats, guinea pigs and chicks that had been fed diets deficient in one or another component of the B complex. The rats and guinea pigs had been obtained from Prof. Herbert M. Evans' Institute of Experimental Biology, University of California, and the chicks from Dr. T. H. Jukes, of the division of poultry husbandry of the College of Agriculture, University of California, at Davis, Calif. The ears of these animals were studied histologically by Dr. W. P. Covell, working under the Coleman grant at the George Williams Hooper Foundation for Medical Research, University of California Medical School. His studies covered not only B_1, riboflavin B_6, the Jukes filtrate factor (pantothenic acid) preventing dermatitis in chicks and the Morgan gray hair factor but also deficiencies in vitamins A, C, D and E (Evans' antisterility factor). No complete studies were made on blacktongue in dogs (pellagra). Striking changes in the eighth nerve were found, especially in those animals which were chronically deficient.

Hogan,[2] describing the changes associated with riboflavin deficiency, said:

> The neurological changes are described as (a) marked degeneration of the medullary sheaths and axis cylinders of the peripheral nerve, (b) fairly common degeneration of the medullary sheaths of the posterior nerve roots with less frequent involvement of the anterior roots of the cord, and (c) degeneration of the medullary sheaths and axis cylinders of the posterior columns of the spinal cord.

This statement is particularly interesting, because the late Albert Gray,[3] studying a case of extreme deafness, found such a picture of the

1. Selfridge, G.: Eighth Nerve High Tone Deafness from a Nutritional Standpoint, Ann. Otol., Rhin. & Laryng. **46**:93-118 (March) 1937.

2. Hogan, A. G.: Riboflavin: Physiology and Pathology, in The Vitamins, Chicago, American Medical Association, 1939, p. 282.

3. Gray, A. A.: Otosclerosis Problem, Including Reports of Two Cases Pathologically Examined, J. Laryng. & Otol. **49**:629-663 (Oct.) 1934.

eighth nerve and then ventured the suggestion that the primary cause of deafness in otosclerosis is an involvement of the nerve and that the bone changes are secondary.

Evidence presented by various other authors who have been studying animals and human beings deficient in the different fractions of the vitamin B complex, particularly subjects with pellagra, shows that all the fractions have a definite influence on the central nervous system and the peripheral nerves as well as on skin lesions. The early reports claimed that nicotinic acid was a specific remedy for pellagra, but it has since been found that thiamine hydrochloride, riboflavin and B_6 are needed, and it now looks as though pantothenic acid and the Morgan factor may be necessary additions before perfect relief is obtained.

Hence, it is obvious that the student should not direct his interest solely to thiamine hydrochloride or to nicotinic acid but should consider the entire vitamin picture.

I have stated repeatedly that neither thiamine hydrochloride nor nicotinic acid can provide complete nutritional protection against eighth nerve and conduction deafness but that other factors of the vitamin B_2 complex as well as other vitamins (A, C, D and E) are equally important. I have also stated that deafness appears to be linked with the various factors related to growth, i. e., glandular products, vitamins, mineral salts (the electrolytes) and amino acids.

Figures 1 and 2 show animals and chicks deficient in vitamins. I shall list the obvious manifestations of deficiencies in these different vitamins and discuss the amounts of vitamin, especially of the vitamin B complex, necessary to keep the deficient animals in good health. I shall compare these with the formulas put out by certain pharmaceutic houses for human beings and shall briefly relate my observations regarding eighth nerve and conduction deafness.

Ever since vitamins have been in use, there has been a great deal of confusion regarding the curative dose in certain diseases, although Cowgill [3a] has written at length on the minimal requirements for man, particularly with respect to the vitamin B complex. The curative dosages for several diseases of man have only recently been understood, in consequence of the pioneering studies of such men as Spies, Jolliffe, Dann, Sebrell, Elvehjem and others.

MANIFESTATIONS OF DEFICIENCY DISEASES

Vitamin A (the Anti-Infective Vitamin).—Among the evidences of vitamin A deficiency are: xerophthalmia, night blindness, photophobia,

3a. Cowgill, G. R.: The Vitamin Requirements of Man, J. Am. Dietet. A. **13**:195-214 (Sept.) 1937.

difficulty in reading at night, momentary blurring while reading;[4] metaplasias of the conjunctiva and cornea; involvement of epithelial cells followed by keratinization in the eye, ear, nose and elsewhere in the body; abscesses in the ear, mucous membranes and elsewhere; keratinization of the skin, principally on the forehead, arms and legs, and myelin changes in the eighth nerve and elsewhere.[5]

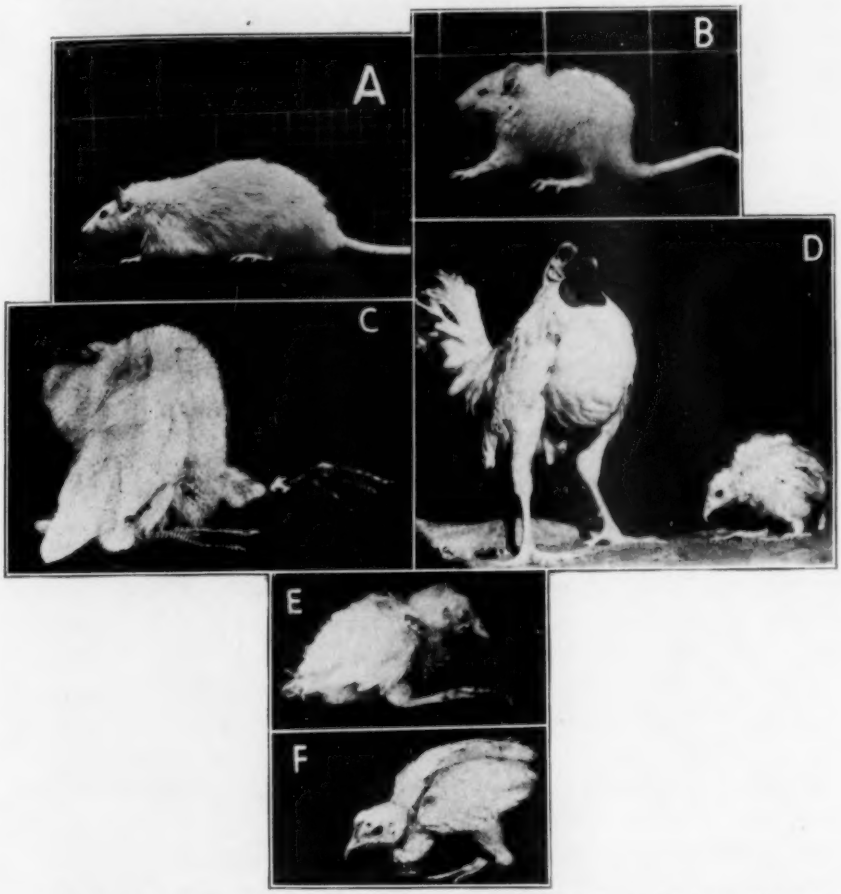

Fig. 1.—*A*, a normal rat. *B*, a rat with acute vitamin B_1 deficiency. His posture indicates an impending attack of vertigo. *C*, a chicken with vitamin B deficiency (polyneuritis). Note the typical retraction of the head. *D*, a rooster (left) which has been given injections of riboflavin and a chick (right) with riboflavin deficiency. *E* shows the effect of pantothenic acid deficiency and *F*, the effect of riboflavin deficiency in chicks. (Photographs *E* and *F* are published by permission of the poultry department of Cornell University.)

4. Cordes, F. C., and Harrington, D. O.: Asthenopia Due to Vitamin A Deficiency, Am. J. Ophth. **22**:1343-1354 (Dec.) 1939.

5. Mellanby, E.: The Experimental Production of Deafness in Young Animals by Diet, J. Physiol. **94**:380-398 (Dec.) 1938.

The principal food sources of the vitamin are milk and milk products, egg yolk, yellow vegetables such as carrots, fresh apricots, green leafy vegetables, green peppers, asparagus, tomatoes and certain fish livers.

Vitamin B Complex.—Thiamine (Vitamin B_1): Manifestations of a deficiency of vitamin B_1 are loss of appetite, anorexia, gastrointestinal changes, hypotonicity, constipation or diarrhea, disturbance of water balance, polyneuritis (beriberi), bradycardia, enlargement of the right side of the heart, vertigo, increased nystagmus (in rotated rats), degeneration of the eighth nerve, spinal cord and other nerves, edema and serous effusion. This factor is necessary for the metabolism of carbohydrates. The principal food sources are liver, yeast, many vegetables, fruits, cereal germs and bran, whole grains, beans and nuts.

Riboflavin (Vitamin G [B_2]): Signs of riboflavin deficiency referable to the eyes are ocular changes,[6] itching and burning of the conjunctiva, photophobia, dimness of vision in poor light, ciliary congestion, keratitis and related changes, as shown by slit lamp examination.

The lips are reddened and shiny. There are macerating and fissuring in the angles of the mouth. A roughening or sharklike appearance of the skin is often observed. Seborrheic accumulation in the nasolabial folds and probably baldness due to seborrhea of the scalp develop. The tongue is reddened, purplish red or magenta rather than the bright red associated with pellagra; the papillae of the tongue are flattened and mushroom in shape rather than atrophic.[7]

The principal food sources of vitamin B_2 are yeast, liver, kidney, wheat germ, lean meat, leafy vegetables (beet tops, kale, spinach), broccoli, fresh beans, peas, carrots and milk.

Nicotinic Acid (Pellagra-Preventive Factor): Persons with pellagra complain of loss of weight, strength (fatigue) and appetite and of symptoms of functional gastrointestinal disorders (dyspepsia, diarrhea or constipation). The under surface and sides of the tongue become fiery red. The patients have so-called neurasthenia, abdominal pain, burning sensations in various parts of the body, numbness, nervousness, palpitation, vertigo, soreness of the lips and tongue, symmetric disturbance of the skin on the face and arms and elsewhere, sometimes with dryness and scaliness, and cutaneous photosensitivity. Mental hebetude, psychoneurotic syndromes, emotional disturbances, sleeplessness, headaches and

6. Kruse, H. D.; Sydenstricker, V. P.; Sebrell, W. H., and Cleckley, H. M.: Ocular Manifestations of Ariboflavinosis, Pub. Health Rep. **55**:157-169 (Jan. 26) 1940.

7. Spies, T. D.; Vilter, R. W., and Ashe, W. F.: Pellagra, Beriberi and Riboflavin Deficiency in Human Beings, J. A. M. A. **113**:931-937 (Sept. 2) 1939.

psychosensory disturbances of special senses (ear and eye) are common complaints.[8]

The principal food sources of nicotinic acid are wheat germ, certain yeasts, peanut meal, rice polishings, egg yolk, buttermilk, milk, lean corned beef, lean pork, chicken, cabbage, carrots, peas, collards, kale, tomato juice and turnip greens.

Vitamin B_6: Persons with the vitamin B_6 deficiency syndrome commonly have skin lesions of seborrheic and desquamative types, ulceration of the ears and about the mouth, swelling of the lower parts of

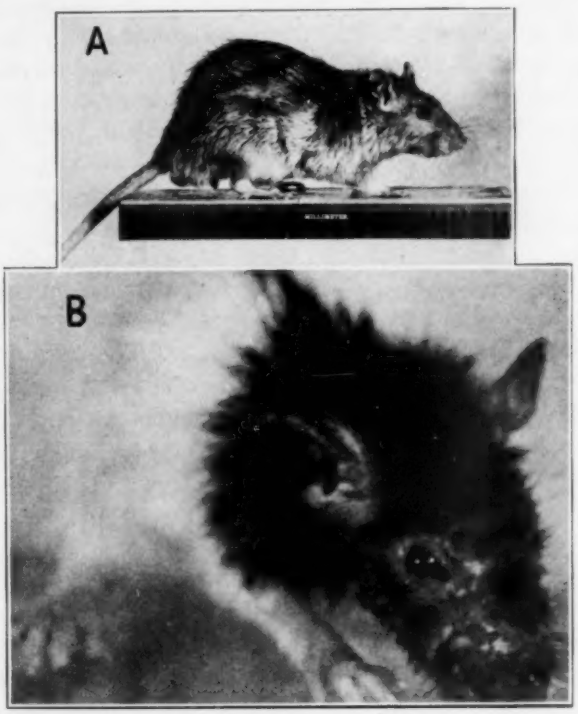

Fig. 2.—*A*, a rat with the deficiency of the Morgan gray hair factor. The animal was grayer than is shown in the picture, which is poorly lighted. *B*, a rat with vitamin B_6 deficiency. Note the ears, mouth and swollen feet.

8. (*a*) Spies, T. D.; Bean, W. B., and Ashe, W. F.: Recent Advances in the Treatment of Pellagra and Associated Deficiencies, Ann. Int. Med. **12**:1830-1844 (May) 1939. (*b*) Frostig, J. P., and Spies, T. D.: The Initial Nervous Syndrome of Pellagra and Associated Deficiency Diseases, Am. J. M. Sc. **199**: 268-274 (Feb.) 1940. (*c*) Spies, T. D.; Walker, A. A., and Woods, A. W.: Pellagra in Infancy and Childhood, J. A. M. A. **113**:1481-1483 (Oct. 14) 1939. (*d*) Aring, C. D.; Evans, J. P., and Spies, T. D.: Some Clinical Neurologic Aspects of Vitamin B Deficiencies, ibid. **113**:2105-2109 (Dec. 9) 1939.

the legs and of the feet and certain types of anemia, especially hypochromic microcytic anemia, which has been produced in dogs. Parkinson's disease has recently been reported to have been cured by large doses of B_6 administered intravenously.

The principal food sources are all cereals, most fruits and vegetables, cane molasses and rice bran. The wheat germ is the richest source.

Pantothenic Acid (Jukes Filtrate Factor Preventing Dermatitis in Chicks): Little is known of the filtrate factor as it may relate to man. A striking thing in chicks with pantothenic acid deficiency, besides the dermatitis, is paralysis of the legs below the hock joints and ability to move only in a backward direction. Jukes [9] said in a personal communication: "Its (pantothenic acid) use may be indicated in various nervous disorders which do not respond to other members of the complex." It is believed that this acid prevents atrophy of cells in the adrenal cortex and probably of cells of the gonads and influences the ratio of potassium and sodium chlorides in the blood and urine. It may influence greatly the postinfluenzal fatigue.

The principal food sources are liver, brewers' dried yeast, egg yolk, liver meal, eggs, cane molasses, broccoli leaves and blossoms, peanut meal, whey, buttermilk, skim milk, whole milk, sweet potatoes, zucchini squash, lean beef, kale, canned salmon, wheat bran, rice bran, canned pumpkin, split peas, tomatoes, corn, peas, soybean meal and cottonseed meal.

Morgan [10] Gray Hair Factor: Any human being whose hair is beginning to gray should have a careful nutritional study, directed mainly to deficiency of all the vitamins, especially the B complex. Animals with advanced deficiency appear very old; some have unhealed ulcers in certain parts of the body.

Histologic studies show atrophy of the gonads and adrenal cortex and changes in the nervous system. The lack of this fraction may explain aging in man. Apparently, a certain reddening of the skin and some types of eczema, particularly in gray-haired people, can be associated with this vitamin.

The principal food sources are rice bran, extracts of liver, certain brands of yeast, cane molasses, vegetables and whole grains. There is probably a little in fruits.

Vitamin C.—Evidences of deficiency of vitamin C are bleeding from the nose, especially in children; petechial spots in various parts of the body; swollen and bleeding gums, indicative of fragile blood vessels, which are often an early sign; changes in the structure of teeth and bones (decalcification), and profound anemia, due to destruction of blood-forming cells in the bone marrow. Many patients have nasal allergies.

9. Jukes, T. H.: Personal communication to the author.
10. Morgan, A. F.: Personal communication to the author.

Definite changes in the middle ear and adhesive deafness are apparently due to lack of vitamin C, the intercellular cementum.[11]

The principal food sources are oranges, grapefruit, tangerines, tomatoes, fresh strawberries, green peppers, fresh cabbage, spinach, brussels sprouts, kale, broccoli, asparagus, turnip tops, beet tops, dandelion tops, apples, bananas, pineapples, green beans, green peas, lettuce, escarole, onions, turnips, parsnips and cauliflower.

Vitamin D.—A deficiency of vitamin D is manifest in various deformities of the bones. Rickets, showing itself as bow legs and knock knees (evidence shown in roentgenograms) is particularly common.

The principal food sources of vitamin D are natural foods of animal origin; fish, salmon, sardines, herring, eggs and milk fat. The most important source is the livers of certain fish.

Vitamin E (Antisterility Factor).—Aside from sterility the outstanding effect of vitamin E deficiency is a general dystrophy of the muscles accompanied by a marked degenerative involvement of the central nervous system and peripheral nerves. Amyotrophic lateral sclerosis is a frequent finding. Atrophy of the stapedius and tensor tympani muscles has been observed by Covell.[12] Alteration in the position of the membrana tympani may be due to a deficiency of this vitamin.

The principal food sources are whole wheat, whole corn, unpolished rice, lettuce and cottonseed oil.

DIETARY CONSIDERATIONS IN TREATMENT

In any one failing to eat enough of the essential food factors for any considerable length of time, one or more deficiency diseases, probably of a subclinical type but often of a very serious type, are apt to develop. Examples are: beriberi (due to excessive use of polished rice), pellagra (in the Southern states it is due to a diet largely of "hog, hominy and syrup," no fruits or vegetables), subclinical scurvy (due to lack of vitamin C), night blindness (lack of vitamin A), Parkinson's disease (related to vitamin B_6) and atrophy of the adrenal cortex and probably of the gonads (due to lack of pantothenic acid). Some of these conditions are definitely related to the use of white flour and cane sugar products.

Experts in nutrition consider the following to be the essential factors in an adult's daily diet: (1) at least 1 pint (473 cc.) of milk or buttermilk; (2) one egg; (3) one leafy salad—such as lettuce, kale or cabbage—and (4) orange, grapefruit or lemon.

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11. Semenov, H.: Some Observations on the Histopathology of Inflammation in the Eustachian Tube and Middle Ear, Tr. Am. Laryng., Rhin. & Otol. Soc. **42**: 563-598, 1936.

12. Covell, W. P.: Personal communication to the author.

Liver, yeast, germ of wheat, rice and other cereal grains and cane molasses are listed as containing a sufficient amount of the B complex, especially of the B_2 factor. Some of these foods are not found frequently in the daily intake of the average human being. Hence, it is not at all surprising that deficiencies of the severe type, involving the general nervous system and the peripheral nerves, are being encountered with greater frequency.

The dosage of vitamin B complex necessary to keep chicks, rats and dogs in a state of health and to promote growth is given in table 1.

The large doses of vitamin B_6 and nicotinic acid are for persons severely ill with pellagra. Moderate doses, 396 mg. to 660 mg., are

TABLE 1.—*Requirement of Vitamin B Complex for Chicks, Rats and Dogs* *

	Thiamine (B_1)	Riboflavin	B_6	Pantothenic Acid	Nicotinic Acid	Gray Hair Factor
Chick (per Gm. of food. [Chicks eat 10-20 Gm. per day])	1.5 micro-grams	2.7 micro-grams	3.1 micro-grams	14 micro-grams	Probably not needed	
Rat (per day).....	10 to 15 micro-grams	10 to 20 micro-grams	10 micro-grams	10 to 150 micro-grams	Probably not needed	
Dog (per Kg. of body weight)	100 micro-grams	100 micro-grams	100 micro-grams	1 mg.	1 mg.	Equivalent to 16 Gm. yeast strain 'L', Anheuser-Busch, Inc.
Man (per day)						
Normal.....	1 mg.	1 mg.	1 mg.	10 mg.	15 mg.	
Curative.....	10 to 40 mg.	5 mg.	2 to 50 mg.	20 to 100 mg.	132 to 500 mg.	

* 1 microgram = one-millionth part of a gram.
2.5 micrograms = 1 Sherman-Chase unit (B_1).
3 micrograms = 1 international unit (B_1).
14 micrograms = 1 chick unit.
1 mg. = 333 international units (B_1).
3 mg. = 1,000 international units (B_1).
9 mg. = 3,000 international units (B_1).

necessary for persons whose urine gives a 4 plus reaction for porphyrin. It takes six to fourteen days to clear the urine of these persons of porphyrin. Larger doses would shorten the time. Spies [12a] reported the intravenous administration of 50 mg. of vitamin B_6 but does not suggest that dose for ordinary use.

It must be understood that basic metabolism differs to a considerable extent in animals, chicks and man, but it is obvious that much is yet to be done in the study of the vitamin B complex as it relates to man. For example, representatives of one pharmaceutic house were asked how many of their capsules were necessary to cure alcoholic pellegra, and their answer was, thirty per day. Yet 50 mg. of nicotinic acid has been given intravenously each hour for ten hours in severe pellagra!

12a. Spies, T. D.; Bean, W. B., and Ashe, W. F.: Note on Use of Vitamin B_6 in Human Nutrition, J. A. M. A. **112**:2414-2415 (June 10) 1939.

My use of California rice bran vitamin B complex syrup was undertaken on the advice of Dr. Samuel Lepkovsky, who was then connected with Evans' Institute of Experimental Biology. He considered it the best preparation on the market, and it was readily obtained. This preparation was used over a period of two years, and in many cases the results were striking, especially in young adults. This preparation contains all the known fractions of the B complex but is not as rich in

TABLE 2.—*Amounts of the Various Factors in Some Commercial Preparations of Vitamin B Complex*

Pharmaceutical House	Name of Preparation	Thiamine (B_1)	Riboflavin	B_6	Filtrate Factor	Nicotinic Acid	Gray Hair Factor
Abbott Laboratories	B complex syrup (prophylactic dose 8 cc.); each 5 cc. contains.....	1.5 mg.	0.6 mg.	0.25 mg.	....	6 mg.	..
Lederle Laboratories	B complex capsules; 1 capsule contains...	1 mg.	0.25 mg.	130 micrograms	12 rat growth units	2 mg.	..
Eli Lilly & Co.	Betalin compound; 1 capsule contains...	1 mg.	40 Sherman-Bourquin units	Not known but 2% concentration will protect rat from lesions ('W' is same as B_6?)	Excess 33 Jukes units per Gm.*	Not definite but thought to be high	..
Parke, Davis & Co.	Combex kapseales; 1 capsule contains...	1 mg.	100 Sherman-Bourquin units	130 micrograms	140 micrograms	5 mg.	..
E. R. Squibb & Sons	B complex vitamin syrup (the rice bran syrup made in Calif.; same as galen B); each 15 cc. contains.....	(2.25 mg.) 750 international units	150 micrograms	1,500 micrograms	375 Jukes units	Rich source (amount not stated)	..
E. R. Squibb & Sons	B complex capsules; 1 capsule contains...	(0.9 mg.) 300 international units	150 micrograms	48 micrograms	14 Jukes units	5 mg.	Unknown

* Eli Lilly & Co. has made no attempt to explain what the filtrate factor is. Probably the Jukes dermatitis factor is included with the Morgan gray hair factor.

some components as certain strains of yeast developed by G. F. Siemers, of Anheuser-Busch, Inc., of St. Louis.

When synthetic preparations of thiamine hydrochloride, riboflavin and nicotinic acid were first available, they were tried singly, to observe which of the three produced the best results. At present nicotinic acid seems to increase the hearing curve better than thiamine. In only a single instance did riboflavin produce a fairly marked change. However, now that preparations of riboflavin are obtainable in stronger potency, a repetition of its use may show greater benefit. Hogan [2] has observed marked changes in the peripheral nerves as a result of riboflavin deficiency.

At present I use pantothenic acid in treating a series of patients who are also taking thiamine hydrochloride and nicotinic acid. This treatment will be carried on over several months, when vitamin B_6 (just recently synthesized) will be added. When the Morgan gray hair factor is available, it will be used in the same way. Spies and others have followed the same procedure in the treatment of patients with pellagra. Ultimately there will be a reversion to the use of a total complex, if and when a preparation is available that contains a maximum dose of each of the fractions.

There is little doubt in my mind that through laboratory studies of the blood and urine it will be found that other vitamins are implicated, as shown in case 1.

OTHER THERAPEUTIC STUDIES

Prostigmine.—For several months I have treated a series of deaf patients, including those with nerve deafness and those with mixed deafness (conduction plus nerve), with prostigmine, injected hypodermically. The use of this material was first suggested to me by Dr. R. D. Shaner, of Hoffmann-LaRoche, Inc., Nutley, N. J. This was prior to the appearance of an article by Davis and Rommel [13] on the treatment of deafness and nervous disorders with prostigmine. I did not understand then, nor is it clear to me now, how prostigmine works—if it does. The drug is a vasodilator and is supposed to liberate esterases rapidly at the neuromuscular junction. Prof. H. H. Beard (biochemist, Louisiana State University and Agricultural and Mechanical College) told me that he had observed dramatic results following its use in myasthenia gravis.

Evidence is not obtainable that prostigmine produces decalcification of bone, nor is it clear what its direct action is on peripheral nerves—whether through its vasodilator effect better nourishment is developed, thus apparently improving the hearing for certain notes of the tone scale, I cannot say. (At a later date I shall show lantern slides of the hearing curves of patients who have been treated with this material over a ten month period.)

It was suggested by Professor Beard that there was a possibility of atrophy of the muscles of the ear. Therefore, Beard and Main, working in the Harriman Research Laboratories of the Southern Pacific General Hospital, undertook the determination of the creatinine and creatine contents of urine specimens from 150 patients with nerve and conduction deafness. Forty-five per cent of the patients showed an output of creatine of over 20 mg. in twenty-four hours. Females normally show no increase.

13. Davis, T. C., and Rommel, J. C.: Treatment of Deafness and Nervous Disorders with Prostigmine, Arch. Otolaryng. **29**:751-758 (May) 1939.

Synthetic Vitamin E.—Since the discovery by Prof. Herbert Evans of extensive muscular dystrophy in vitamin E–deficient rats and the extension of this discovery by Covell [12] to include the tensor tympani and stapedius muscles, it looks as though the use of alpha tocopherol (synthetic vitamin E) may prove of great value, especially since histologic studies have shown marked degeneration of peripheral nerves and the general nervous system in animals with vitamin E deficiency. I am using vitamin E in tablet form, under the trade name of ephynal,[14] in the treatment of a series of patients. The use of this preparation will be continued for several months. Some of the patients have already had a series of injections of a solution of prostigmine methylsulfate. A full report on its use will be made at a later date. In some cases tinnitus is influenced satisfactorily and the hearing is improved somewhat.

Androgen.—Through the aid of Dr. Max Gilbert, of the Schering Corporation, Bloomfield, N. J., I have been able to begin the trial of androgen (in the form of oreton) in several women of different ages, some of whom had been given estrogens previously. A report of these cases has been published.[15]

The basis for the use of this androgen was an article by Bernstein and Gillis [16] on the treatment of otosclerosis with "sex hormones." These authors reported apparently satisfactory results in a number of cases, but their tests were confined to the hearing of voice. No audiometer was used. Their treatment included the administration of 10 mg. parenterally once a week for three months. At present I give 10 mg. twice weekly, and I shall probably continue the treatment over a longer period.

Vitamin C.—A recent study of the blood of persons who had eaten no citrus fruits or juices or tomato juice for the thirty-six hours preceding the test showed that in every case except 2 the value for vitamin C was below 0.6 mg. per hundred cubic centimeters.

This study appears to be an important one, because vitamin C is the intracellular cementum, and its lack causes bone to decalcify and has to do with the formation of adhesions. Vitamin C deficiency is related also to failure of the resorption of mesenchyme and produces, as Covell has observed, intracellular edema throughout Corti's organ and also changes in the eighth nerve.

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14. The preparation was made available by Dr. R. D. Shaner, of Hoffmann-LaRoche, Inc., Nutley, N. J.

15. Selfridge, G.: Chronic Progressive Deafness with Special Reference to Estrogenic Substances: Further Contribution, Ann. Otol., Rhin. & Laryng. **49**: 52-75 (March) 1940.

16. Bernstein, J., and Gillis, L.: Otosclerosis Treated with Sex Hormones, Lancet **2**:1368-1369 (Dec. 30) 1939.

REPORT OF CASES

The following case reports suggest the truth of Wolbach's [17] statement: "No one vitamin is concerned in the maintenance of myelin." This statment should perhaps read as follows: All vitamins are involved in the maintenance of optimum health and all are equally necessary in many, if not all, disease conditions of nutritional origin involving the central nervous system and peripheral nerves.

CASE 1.—Miss L. S., aged 17, was seen on Aug. 14, 1939, with the presenting complaint of deafness in both ears.

History.—Deafness had been noticed in infancy. There was no history of earache. The mother and grandparents were deaf. In regard to allergy, there was no history of hay fever, asthma or hives. The teeth were good. In childhood the patient had 1 pint (473 cc.) of milk daily, no eggs, cereals, leafy salads, oranges or cod liver oil but a lot of bread, potatoes and candy. In adolescence she had plenty of fruits (except citrus), vegetables, salads, milk and butter. In spite of a seemingly normal diet she was deficient in vitamins A, B and probably C. She had had measles but no other childhood diseases. She had also had a mild attack of influenza. She had a history of frequent colds. For the past year, especially during school hours, she had often become shaky, nervous and unable to control herself (probably some emotional instability). As a result, her pulse rate would jump to 100. Catamenia was normal.

Physical Examination.—The patient's general health was found to be good. She was not constipated. Her weight was 108 pounds (49 Kg.) and her height 5 feet 2 inches (157 cm.). Her nose was normal. Her tonsils and adenoids had been removed. When testing was done with a tuning fork, the sound was scarcely heard by bone conduction. On the forehead, an area of skin 2 by 4 inches (5 by 10 cm.) showed keratosis, and there was some above the left breast and on the right cheek but none on the arms and legs.[18]

Laboratory Data.—The basal metabolic rate was minus 90 per cent; the pulse rate at rest was 76. On August 17 the hemoglobin content was 75 per cent, the red blood cell count 4,450,000 and the white blood cell count 7,850. The vitamin C content of the blood was 0.625 mg., the calcium content 9.2 mg. and the phosphorus content 4.7 mg. per hundred cubic centimeters. The urine (24 hour specimen) contained 3.4 international units of vitamin B_1 (very low). The urine gave a 1 plus reaction for porphyrin. The total creatinine was 0.491 mg. per hundred cubic centimeters (preformed creatinine, 0.483; creatine, 0.008).

A dextrose tolerance test gave the following results:

	Dextrose Content of Blood (Mg.)	Urine Specimen	
		Sugar	Acetone
Before administration of dextrose.....	76.6	0	0
1 hour after dextrose was given.....	80.2	0	0
2 hours after dextrose was given.....	90.0	0	0
3 hours after dextrose was given.....	60.2	0	0

17. Wolbach, S. B.: The Pathologic Changes Resulting from Vitamin Deficiency, J. A. M. A. **108**:7-13 (Jan. 2) 1937.

18. Lehman, E., and Rapaport, H. G.: Cutaneous Manifestations of Vitamin Deficiency in Children, J. A. M. A. **114**:387-393 (Feb. 3) 1940.

Roentgen Examination.—The chronologic age of the patient was 17 years and 3 months; the bone age as revealed by roentgenograms was approximately 17 years. There was no roentgen evidence of former rickets.

Treatment and Course.—On August 14 the administration of a liver and stomach extract with iron in capsules (Lextron), three times a day, was instituted for

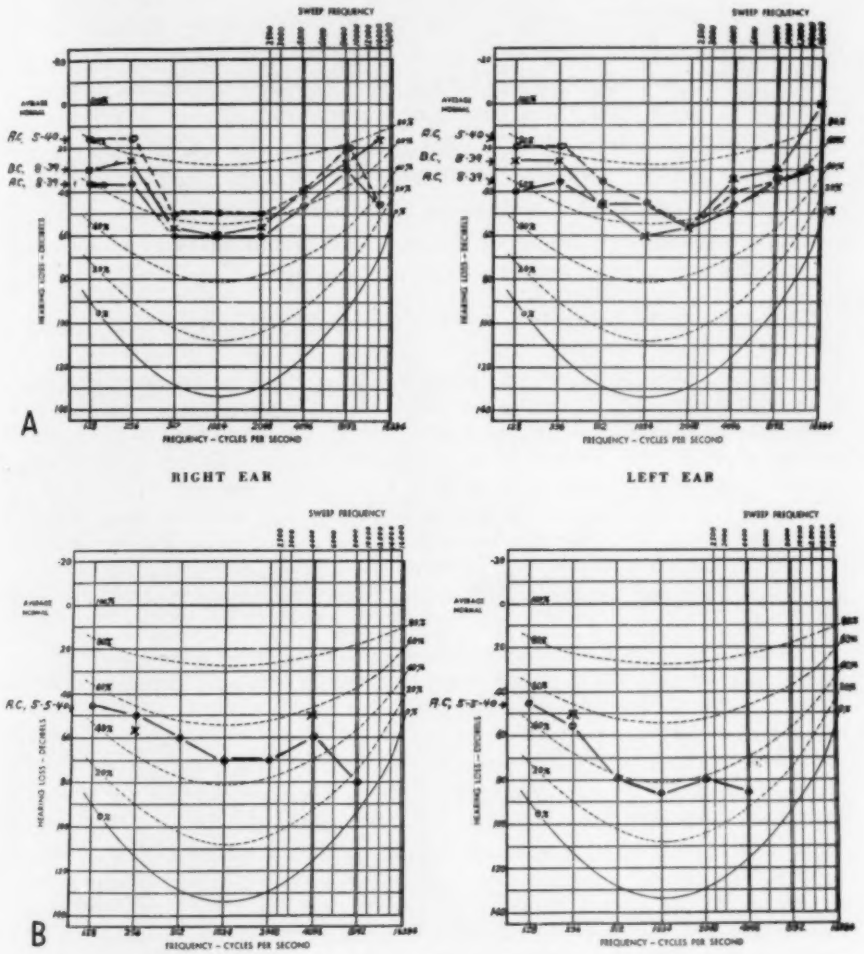

Fig. 3.—Audiograms (*A*) of the patient in case 1 and (*B*) of her mother (nerve deafness).

anemia. On Jan. 1, 1940 the hemoglobin content was 82.5 per cent, the red blood cell count 4,540,000 and the white blood cell count 8,300. The administration of this liver and stomach extract was continued. The intake of foodstuffs rich in vitamin A was increased and carotene, 10,000 U. S. P. units three times a day, was given for the keratosis. On February 10 a marked improvement of the keratosis and a gain in weight of 12 pounds (5.5 Kg.) were noted. The administration

of the liver and stomach extract and carotene was continued. On April 6 ascorbic acid (300 mg. daily) was added to the liver and stomach extract and carotene. On May 31 the administration of the liver and stomach extract was stopped; that of carotene and ascorbic acid was continued, with the addition of thiamine hydrochloride (3 mg.) and nicotinic acid (66 mg.) night and morning.

The question at once arises, Can the dip shown in the audiogram between 256 and 4096 cycles in both ears be due to an increased amount of hyperplastic bone as the result of vitamin A deficiency in certain areas? With that idea in mind, I wrote to Edward Mellanby as follows: "Have you found that the addition of carotene or vitamin A produces decalcification of certain areas?" His reply, dated April 8, is as follows: "I have carried out experiments on this point, and the tissues are being prepared for their final examination. I should think an answer to this question will be forthcoming in the course of a few weeks. If I have any results worth reporting, I shall write you again."

CASE 2.—Miss K. B., aged 17, was seen on Oct. 13, 1939 with the presenting complaint of deafness in both ears.

History.—Deafness was noticed when the patient was around the age of 8. It apparently followed colds and abscesses of the ears during the previous years. There was no other deafness in the family. There was no history of hay fever, asthma or hives. The teeth were good. In recent years the patient's diet had been very poor. It was deficient in vitamin A and several fractions of the vitamin B complex. She had had all the usual children's diseases and one severe attack of bronchopneumonia. Catamenia began at 13 years and was fairly regular until the age of 16, when it stopped. It began again when the patient was given thyroid. (The basal metabolic rate at this time was between —29 and —32 per cent.)

Physical Examination.—The general health was found to be good. The patient smoked a great deal. Her weight was 115 pounds (52 Kg.). Her height was 5 feet 3 inches (160 cm.). Her nose showed a slight deviation of the septum to the right. Her sinuses were normal. Her tonsils had been removed. There was some lymphoid tissue in Rosenmüller's fossae. The right ear was gray, the light spot absent and the drum membrane movable. I was unable to see the anterior wall of the left external canal.

Laboratory Data.—The basal metabolic rate was between —29 per cent and —32 per cent. The urine (twenty-four hour specimen) contained 24 international units of vitamin B_1. It gave a 4 plus reaction for porphyrin. The total creatinine content was 1.61 Gm. per hundred cubic centimeters (preformed creatinine, 1.22 Gm.; creatine, 0.39 Gm.). The blood contained vitamin C, 1.07 mg., sodium, 299 mg., and potassium, 18.6 mg. per hundred cubic centimeters.

Treatment and Course.—On October 13, treatment with aminoacetic acid was instituted, 1 flat teaspoonful (15 cc.) being given after each meal. On Jan. 22, 1940 the patient directed my attention to cracks in the skin at both sides of the mouth. On February 17 the administration of aminoacetic acid was stopped and treatment with thyroid, riboflavin and nicotinic acid was instituted. On May 21 the administration of riboflavin was stopped, while that of thyroid and nicotinic acid was continued, with the addition of a preparation of the B complex in capsules (E. R. Squibb & Son) and vitamin E.

This case is one of adhesive deafness, probably with atrophy of the tensor tympani muscle and perhaps also of the stapedius muscle rather than otosclerosis. The lack of luster, the loss of the light reflex and the loss of motion of the posterior half of the drum membrane are suggestive of this. The increase of creatine in the urine in the absence of evidence of atrophy of muscles other than the tensor tympani and the stapedius and recent work of Mackenzie and McCollum [18] on the cure of muscular dystrophy by alpha tocopherol and the effect of this substance on creatine metabolism appear to support such a conclusion. This idea has been further supported by Covell's observation of atrophy, especially of the tensor tympani muscle, in Prof. Herbert M. Evans' vitamin E–deficient rats.

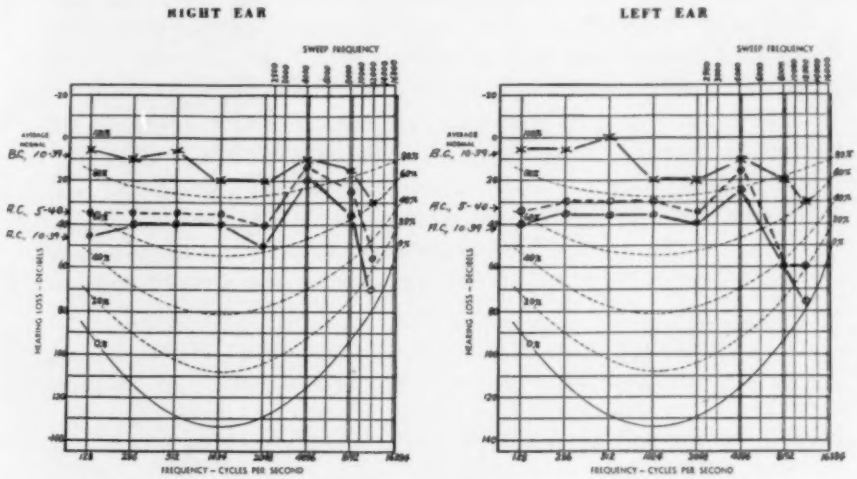

Fig. 4.—Audiograms of the patient in case 2.

Cracks in the skin at the sides of the mouth are considered by Sebrell and Spies as positive evidence of riboflavin (vitamin G, or B_2) deficiency.

The patient's history of frequent colds and aural abscesses before the age of 8 points to a vitamin A deficiency, and the finding of porphyrin 4 plus in the urine suggests the use of nicotinic acid in larger doses than are present in the vitamin B complex preparation which she has been taking. The low basal metabolic rate tells a further story of her general metabolic disturbance.

18. Mackenzie, C. G., and McCollum, E. V.: The Cure of Nutritional Muscular Dystrophy in the Rabbit by Alpha-Tocopherol and Its Effect on Creatine Metabolism, J. Nutrition **19**:345-362 (April) 1940.

SUMMARY AND COMMENT

This paper is the result of a request from certain friends, who recently asked me to tell what studies of deafness I was engaged in. I have thought the general subject of nutritional deficiency, especially in regard to vitamins, might be simplified if the obvious signs and symptoms of deficiencies in human beings were briefly described and if the principal food sources of each vitamin were given. I have thought, too, that by thus simplifying the subject one might avoid duplication of expensive laboratory studies and perhaps create a greater interest on the part of otologists in the fascinating subject of nutrition.

Two cases have been described and are presented in detail only to show the complexity of the subject and to point out again what I have stated repeatedly, that deafness is not caused by any one thing but is related to all the factors involved in growth, that is, endocrine products, electrolytes, amino acids and vitamins.

It is becoming increasingly evident that time is the great factor in the treatment of chronic progressive deafness (nerve and conduction). Physicians as well as patients must realize that if improvement is to result treatment may have to be followed for years rather than for weeks or months, as most expect. Indeed, as in many other chronic diseased states, one is fortunate if the progress of the disease can be checked. Complete cure of chronic disease is perhaps beyond the realm of probability.

It is also becoming increasingly evident that so far as the vitamin B complex is related to diseased conditions, especially those of the general nervous system and particularly those of the peripheral nerves, the present known factors ultimately will be combined in more potent amounts then they are at present in products put out by the pharmaceutic houses in this country.

More evidence is accumulating that the time to begin the prevention of deafness is really six months before conception. Each parent should be taught that an optimum diet, containing the essential food factors (vitamins), is necessary in order to beget a healthy normal child and that such a diet should be carried out by the pregnant mother. The same careful supervision should be given to the diet of the child through infancy, babyhood, childhood and adolescence.

384 Post Street.

DIAGNOSIS AND DIRECT LARYNGOSCOPIC TREATMENT OF FUNCTIONAL APHONIA

WILLIAM A. LELL, M.D.
PHILADELPHIA

Functional aphonia may be defined as a clinical entity in which there is sudden complete loss of voice without any demonstrable organic lesion in the neuromuscular apparatus used in voice production. In cases in which there is not complete loss of voice, a barely audible whispering note may be the only result of attempts at phonation. The condition may be precipitated without any forewarning, and the patient may retire in full possession of the voice only to awaken and find vocalization impossible. Likewise, after the condition has persisted for a variable time, the vocal powers may return as suddenly and as mysteriously as they disappeared, only too often, however, to be lost again.

E. C. MacLeod,[1] in 1930, reporting her clinical observations in the treatment of a large number of patients, offered to differentiate two types of the disease:

1. True aphonia, in which there is little or no approximation of vocal cords on attempts at phonation. In cases of true aphonia the glottis is in the position of normal expiration, or very slightly narrowed.

2. Hyperphonia, in which the cords are brought forcibly together and the ventricular bands are also approximated, making phonation almost entirely impossible. If a voice is produced at all, it is high in pitch and very hard. Occasionally, in patients with aphonia of this type double voice is the result, the vocal cord note being relatively high, while the superimposed note from the ventricular bands is of a much lower pitch.

Although the foregoing classification is based on MacLeod's clinical findings, it does not take into consideration any of the factors which may produce aphonia; consequently, recent observers have preferred to classify functional aphonia as voluntary and involuntary. Under the first, of course, would come the condition of the malingerer, who attempts to simulate the disease in order to deceive. Under the second would fall the true cases of functional aphonia. Among these are found the

1. MacLeod, E. C.: Demonstration of Apparatus Used to Treat Functional Aphonia and Allied Disorders, Proc. Roy. Soc. Med. (Sect. Laryng.) **23**:25-26 (April) 1930.

cases of highly emotional, unstable and susceptible persons, in whom the condition may be precipitated by mere fright, shock and anger or may be secondary to an infection of the upper respiratory tract or some other focus of infection not directly related to the phonatory apparatus.

Mackenzie,[2] after observation of a large number of patients, expressed the belief that the origin of functional aphonia is almost always associated with an inflammatory lesion in the upper respiratory tract, producing laryngitis, and that when the laryngitis clears the patient cannot sufficiently contract the muscles that adduct the vocal cords. The patient catches cold, the larynx becomes congested and hoarseness or aphonia ensues. The congestion soon disappears, but the aphonia persists because of the feeble adductive power of the cords.

Marsh[3] in 1932 reported 16 cases of functional aphonia. In 15 of the 16 cases, foci of infection in the upper respiratory tract were believed to be the direct cause of aphonia. According to his reports, the aphonia cleared up when the foci of infection were eliminated.

Thomson,[4] Moore,[5] Tucker[6] and other clinical observers have persistently noted the presence of an associated inflammatory reaction in the vocal cords or upper respiratory tract in most of their cases of functional aphonia. An analysis of the cases reported in the literature and those encountered in the author's personal experience shows that by far the greater number of patients are females, a rough estimate being a ratio of 7 females to 1 male. As to age, the highest incidence is in persons between 18 and 34.

DIAGNOSIS

The diagnosis of functional aphonia rests on the general emotional background of the patient and both mirror and direct laryngoscopic examination. The laryngeal image should reveal an apparently healthy condition of the laryngeal mucosa, the absence of intrinsic growth and perfect ordering of the nervous laryngeal mechanism except that on attempted phonation both vocal cords begin to approach the median line but fail at a point short of approximation. Having for a moment been almost approximated and the controlling power being lost, they immediately resume the partially approximated position. As in the case of

2. Mackenzie, M.: A Manual of Diseases of the Throat and Nose, London, J. & A. Churchill, 1880, vol. 1.

3. Marsh, F. D.: Functional Aphonia: Some Observations on Its Clinical Aspects, Lancet **2**:289 (Aug. 6) 1932.

4. Thomson, St. C.: Cancer of Larynx, New York, The Macmillan Company, 1930.

5. Moore, W.: Personal communication to the author.

6. Tucker, G.: Inflammatory Tumours of the Vocal Cord: Direct Laryngoscopic Observations, J. Laryng. & Otol. **51**:563-573 (Sept.) 1936.

a hysterical manifestation in any other part of the body, so here the laryngeal findings never form an accurate duplicate of genuine paralysis. Since functional aphonia can counterfeit only those forms of paralysis which can be assumed by voluntary effort, it could hardly simulate adduction of the cords, which is a purely involuntary motion, occurring only during the acts of respiration, phonation and cough.

The hysterical affection may resemble bilateral adductor paralysis of the recurrent laryngeal nerves; in this, however, all the muscles of the larynx are completely paralyzed and the cords are absolutely motionless and in a position midway between extreme adduction and abduction. Besides, bilateral paralysis is rare, and, while in functional aphonia the cords are motionless, they do not occupy the same relative position as in bilateral paralysis. This position of the vocal cords cannot be voluntarily assumed, for the instant that respiration occurs the glottis will be widened and movement can be seen to take place. Aphonia due to mechanical interference with the proper closure of the cords, on account of thickening of the mucous membrane covering the arytenoids or the anterior commissure, may often be confusing, but by careful examination an organic lesion can be excluded.

Thus it may be seen that a close study of the larynx will clear up the diagnosis and enable one to determine that the aphonia is a functional disorder and not due to any lesion. The laryngeal image will not present a complete picture of any of the forms of paralysis. There will also be the accompanying evidences of the hysterical temperament. Invariably, cough is present with functional aphonia, while in paralysis of the adductors it is entirely lost—the possibility of a cough being dependent on the ability to close the glottis.

TREATMENT

Throughout the literature, many methods have been suggested for the restoration of the voice and the apparent cure of these conditions. Only too often the usual preparation for an operation and the actual administration of an anesthetic have been resorted to. Many patients, however, have not been fortunate enough to get away so easily and have been subjected to stimulation from galvanic or faradic shock, which, according to one observer, "should have been enough to make a deaf mute talk."

Among the more humane methods, MacMahon [7] reported the following treatment for complete functional aphonia.

> The patient sits upright in the chair with the mouth opened widely. I put the middle finger of my right hand on the back of the patient's tongue and press it

7. MacMahon, C.: Treatment of Functional Aphonia, J. Laryng. & Otol. **47:** 243-246 (April) 1932.

firmly. I do this for a few seconds and after a short rest repeat the procedure. Sometimes there is a strong resistance at the back of the tongue, but one quick and strong pressure which gives a little pain overcomes the resistance. I then place the fingers of the left hand on the thyroid cartilage and as I depress the back of the tongue and with it the larynx, I press firmly with my thumb and first finger of the left hand on the posterior edges of the thyroid cartilage. At the same time, I ask the patient to cough. If the cough is fairly strong, I tell the patient to cough again and to finish the cough with the sound "ah." Directly the "ah" is heard the patient can begin to speak with vibrations. At first voice may be very highly pitched but with repetition of this procedure it becomes deeper and more resonant and often quite normal in strength.

A falsetto voice has often been employed in reeducation for voice production. In this method the patient is asked to close the mouth tightly and to strike a high note, which will resound in the vault of the nasopharynx. After this practice has been kept up for several minutes, the pitch is lowered gradually, so as to allow the patient to gain confidence in the production of the high tone. This having been accomplished, he can be gradually made to repeat those tones with the mouth open. Now, words can be substituted in the same tone, and he will suddenly realize that he is talking. In this procedure his attention is attracted to the nose rather than to the larynx, and he does not realize that the sounds are really being produced in the latter organ.

Perepel,[8] in a lengthy article on the physiology of hysterical aphonia, enthusiastically transcended into the field of metaphysics for an explanation of the underlying factors in functional aphonia. He reported an interesting case of a girl who responded with complete recovery to psychoanalysis after each attack.

Winkelman,[9] in his report of 5 cases of functional aphonia, attached a great deal of importance to the social background as a factor in bringing about this state. He expressed the belief that the patient assumes aphonia as a defense mechanism, and he cited several cases to prove this point. In his opinion, unless there is a proper readjustment of the disturbing factors in the person's environment, it is impossible to complete the cure of the patient.

Barlow[10] expressed the belief that an unstable and emotional background is the primary etiologic factor in hysterical aphonia.

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8. Perepel, E.: Physiology of Hysterical Aphonia and Mutism, Internat. J. Psycho-Analysis **11**:185-192 (April) 1930.

9. Winkelman, N. W.: Diagnosis and Treatment of Hysterical Aphonia and Diagnosis of Malingering in These Cases, M. Clin. North America **21**:1211-1220 (July) 1937.

10. Barlow, R. A.: Newer Concept of Functional Aphonia, Tr. Am. Laryng. A. **52**:23-34, 1930.

William F. Moore in 1929 reported a case of a woman 35 years of age with a history of periodic loss of voice over six years. The attack usually followed exposure to cold or getting the feet wet. The case was followed over a period of five months, during which time local medication and electric stimulation were tried without result. Because of the lack of improvement and the patient's sense of constriction in the larynx a diagnostic bronchoscopic examination was performed. The result was negative, but the next day the patient had recovered complete use of the voice. In the last six years this patient has had three recurrences of the same condition, and in each instance the voice has been completely restored immediately after bronchoscopic treatment.

Of greatest importance in the treatment of these conditions is the fact that one first must clear up the inflammatory process; this as a rule is sufficient to restore the normal voice, although it may require several weeks. However, in the occasional case in which the condition is more obstinate, one may have to resort to some other methods in order to restore the voice. Direct laryngoscopic examination in these cases is extremely useful, first, because the technic is extremely simple and, second, because it is extremely effective. Of the 5 patients whose cases are here reported, all had to have direct laryngoscopic examination at least once for complete restoration of the normal voice.

In the direct laryngoscopic treatment of so-called hysterical or functional aphonia, the anterior commissure laryngoscope is the instrument of choice. Application of a swab saturated with a 10 per cent solution of cocaine is made in the anterior portion of each piriform sinus. This gives sufficient anesthesia to permit the introduction of the laryngoscope for inspection of the interior of the larynx. The tip of the laryngoscope is introduced into the larynx until the ventricular bands are propped apart and the true cords can be clearly seen. The patient is then requested to phonate, and when phonation is attempted there is sufficient spasm of the true cords from the pressure of the laryngoscope to bring the cords together, and a tone will be produced. The introduction of the laryngoscope must be accurate, so that no trauma to the true cords occurs. When the patient produces a tone, he is asked to repeat it, and the fact that he has produced a tone is emphasized, so as to make a definite mental impression. The laryngoscope is then withdrawn to a point above the level of the ventricular bands, and the patient is again asked to phonate. He is then allowed to sit up on the table and is again requested to phonate. In every case in which this procedure has been carried out, the patient has still continued to phonate after the laryngoscope has been removed. The patient must feel that the voice has been restored by the examination.

This method has been successfully used by Tucker in a series of cases.

SUMMARY AND CONCLUSIONS

In the histories of the cases here reported, the most outstanding etiologic factor has been the presence of an acute inflammatory process, either in the larynx proper or in the surrounding structures. It appears that the inflammatory reaction, particularly when confined to the larynx or the tracheobronchial tree, produces changes in the mechanism of voice production so as to cause aphonia. This process may be evidenced by an acute inflammatory reaction of the cords proper or by an involvement of the ventricular bands producing redundancy of the mucous membrane and thereby interfering with the production of the normal tone. In these cases aphonia persisted after the etiologic factor had been removed.

Before any condition is diagnosed as functional aphonia, careful studies should be carried out to eliminate the possibility of the presence of an organic lesion, especially one in the subglottic region of the larynx. Roentgen examination of the chest is important to rule out the possibility of infection with acid-fast bacilli.

Fig. 1.—Photograph of the larynx (case 1). Note the lack of complete adduction of both cords, particularly in the region of the posterior commissure. This finding was constantly present, although there was no pathologic condition in the larynx proper.

Direct laryngoscopic inspection may be utilized in these obstinate cases not only as a means of examining the subglottic larynx but also as a therapeutic method of getting the patient to phonate and thereby reestablishing his confidence in producing normal tones.

Five cases of particular interest are here reported in detail. In these cases the aphonia persisted after the original infection of the upper respiratory tract had been cleared up. The voice was completely restored by the direct laryngoscopic procedure. Photographs of the larynxes of the patients are also presented.

REPORT OF CASES

CASE 1.—L. B., a white man aged 39, was first seen in 1935 because of hoarseness and a scratchy sensation in the throat. At the time of the first examination he had a barely audible whispering voice. Mirror examination of the larynx revealed that both cords had normal motility except that immediately after complete adduction they would separate again. This condition was present consistently (fig. 1).

The check-up studies gave entirely negative results. The blood was normal. The Wassermann reaction was negative. Roentgen examination of the chest gave negative results. A lateral roentgenogram of the neck did not show any abnormal condition. In going over this man's past history, it was found that he had been receiving a disability pension for his war service and that there was a possibility of this pension being discontinued. Whether this was the predominant factor in his persistent hoarseness it is difficult to say. Direct laryngoscopic examination for treatment revealed both cords to be normal. The subglottic larynx was entirely without abnormality. During the examination the patient was made to phonate, and after the examination he was able to produce a good audible tone. However, the following day he again became aphonic. After he had been treated several times in this manner, he was able to produce a good tone and has been free of symptoms to date.

Case 2.—E. M., a white woman aged 32, was referred by Dr. Norman Lepper because of persistent loss of voice of several weeks' duration following infection of the upper respiratory tract. Although the infection had been cleared, the aphonia persisted. Mirror examination of the larynx revealed the motility of both cords to be normal. However, the mucous membrane was inflamed, and some secretion could be seen in the region of the anterior commissure. Both cords were smooth. The posterior commissure was entirely normal. Both arytenoids appeared normal. The patient was admitted to the Abington Memorial Hospital for study, to rule out the possibility of infection with acid-fast organisms in spite of negative laryngeal findings. Roentgen examination of the chest and a lateral roentgenogram of the neck revealed nothing of importance. The swallowing function was normal. The Wassermann reaction was negative, and the complete blood count was within normal limits. By direct laryngoscopic treatment, reeducation in the use of the voice and reassurance that there were no abnormal changes in the larynx or chest, this patient was enabled to regain complete control of her voice and has retained it to date.

Case 3.—C. T., a white woman aged 30, was referred by Dr. John W. Froggatt because of laryngitis and complete loss of voice. The patient was a singer. The loss of voice had followed immediately after she contracted a cold and was associated with a severe cough. Although the cough subsided, she was still unable to phonate. Mirror examination of the larynx revealed both cords to be acutely inflamed and somewhat thickened, although the entire surface of both cords was smooth. Both ventricular bands approximated before the cords became completely adducted. This in part explained the loss of voice. Roentgen examination of the chest gave negative results. The patient was treated by intralaryngeal instillation of monochlorphenol, but in spite of improvement in the inflammatory reaction of the larynx, she was unable to phonate. When the inflammatory reaction had entirely cleared up, direct laryngoscopic examination was carried out, with subsequent return of the normal voice.

Case 4.—F. S., a white woman aged 28 years, was referred to my service at the Mount Sinai Hospital because of periodic hoarseness and intermittent aphonia. Examination of the pharynx disclosed marked pharyngitis, also extremely infected tonsils. Mirror examination of the larynx revealed the mucous membrane to be

beefy red; both cords were markedly inflamed, although the surface was smooth. On phonation, both cords fell short of complete adduction, and the quality of the voice was a barely audible whisper. Roentgen examination of the chest revealed evidence of peribronchial thickening. Examinations of the sputum gave negative

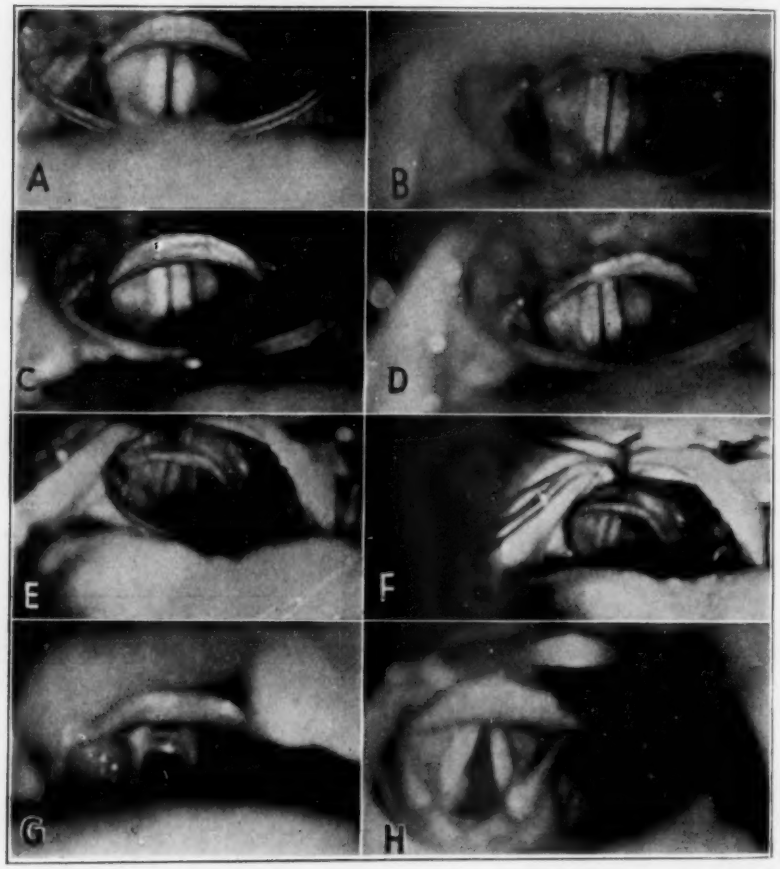

Fig. 2.—Photographs of the larynx. *A* (case 2), during the stage of acute infection of the upper respiratory tract. Note the thickness of both cords, with the presence of secretion beneath the epiglottis. *B* (case 2), after the infection of the upper respiratory tract had subsided. Note the normal appearance of the cords. In *C* (case 3), note the markedly thickened appearance of both cords during the period of the acute inflammatory reaction of the larynx. In *D* (case 3), note the loss of thickening and the return to normal condition of both cords after the inflammatory reaction had cleared up. In *E* (case 4), note the presence of thick secretion beneath the posterior portion of the epiglottis, which in part was the cause of the aphonia. The cords also are markedly thickened. *F* shows the larynx of the same patient after improvement. *G* (case 5), during the period of acute infection of the upper respiratory tract. Note the thick secretion extending from one cord to the other in the anterior portion of the larynx. *H* shows the same larynx after the infection of the upper respiratory tract had been completely cured.

results. The Wassermann reaction was negative. A complete blood count showed an increase in white blood cells; otherwise the blood was normal. The patient was referred to the ear, nose and throat clinic for tonsillectomy and treatment of the sinuses, but in spite of the fact that suspected foci had been cleared up, the aphonia persisted.

Direct laryngoscopic examination revealed the subglottic larynx to be entirely normal. Here again it was noted that the cords were able to adduct but were unable to maintain complete approximation. With the tip of the laryngoscope above the level of the cords, the patient was able to phonate and produce a good tone. This tone was reproduced after the patient returned to her room. However, several days after treatment she again lost her voice. After three direct laryngoscopic examinations, she had completely regained the use of her voice and had not had any recurrence of aphonia to date.

Case 5.—L. S., a white woman aged 24 years, was referred by Dr. Paul Parker because of hoarseness and persistent cough of two weeks' duration. The cough was

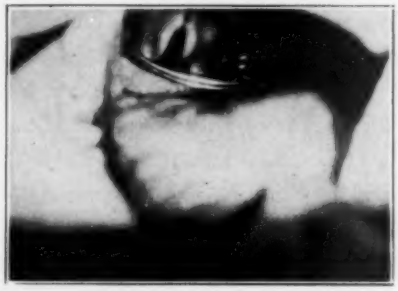

Fig. 3.—Photograph of the larynx of a patient with bilateral vocal nodules producing hoarseness. In this case there is a definite organic background for the aphonia; this type of disease should not be confused with the functional type.

rather productive of thick, tenacious secretion. Mirror examination of the larynx revealed the motility of the cords to be normal. Both cords appeared acutely inflamed and thickened. Both ventricular bands showed marked inflammatory reaction and hypertrophy. In the region of the anterior commissure, thick lumps of mucus could be seen clinging to the mucosa. In going over the history of this patient, it was found that she had been doing nursing duty in a tuberculosis sanatorium. Because of this factor, a roentgenogram of the chest was made, but the results reported were entirely negative. The patient was placed on a regimen of vocal rest and treated medically for the acute tracheobronchitis. However, after the inflammatory reaction of the larynx had subsided, she still was aphonic. After several weeks of persistent attempts at reeducation in the use of her voice, direct laryngoscopic treatment was carried out, and she gradually became able to phonate until her voice returned to normal. In this case, reeducation in the use of her normal voice was accomplished by reassuring the patient that she did not have a tuberculous infection.

SIGNIFICANCE OF THE p_H OF NASAL SECRETIONS IN SITU

NOAH D. FABRICANT, M.D.
CHICAGO

Despite the fact that scientific investigation of the p_H mechanism began more than a half century ago, it is only recently that the interest of practical technologists at large—and rhinologists in particular—has been aroused. The subject of hydrogen ion concentration was poorly defined and lacked a clear, comprehensible nomenclature. With the advent, however, of comparatively new and simple methods for measuring hydrogen ion concentration, it has been possible to awaken the clinician's interest in a type of investigation that has practical application to innumerable and diversified fields. In the realm of medicine, the "p_H influence" is intimately associated with general health. More specifically, in the case of blood it is of paramount importance. The "p_H influence" is not restricted to blood alone, for the p_H of spinal fluid, lymph—in fact all body fluids, including the nasal secretions—has definite clinical significance.

It will be recalled that the p_H scale, a means of expressing hydrogen ion values numerically, is today in common use. The working range of the p_H scale lies between 0 and 14. The midpoint of the range is 7—the p_H of pure water—which is the true neutral point. Values below p_H 7 indicate an increased hydrogen ion concentration, or predominance of the acid reaction, while values above 7 indicate an alkaline reaction.

The value of p_H has been determined in several ways: by use of the hydrogen electrode, by use of the quinhydrone electrode or by colorimetric procedures. These methods are highly involved and subject to considerable error. Further, a factor not without importance is the circumstance that biologic fluids generally are susceptible to changes in p_H almost instantly on exposure to air. This is particularly true of nasal secretions. To offset such a phenomenon, the use of the glass electrode with its

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From the Department of Laryngology, Rhinology and Otology and the Department of Pathology, University of Illinois College of Medicine.

This is part 1 of the work that has received the Casselberry Award of the American Laryngological Association for 1941.

associated measuring equipment has made it possible to measure the p_H of nasal secretions in situ with ease and precision.

Early in the course of my own investigations, I was impressed by the fact that studies of the p_H of nasal secretions accomplished elsewhere involved the collection of samples of nasal secretion either by suction or by having the patient blow his nose. Then too, a frequent procedure was to record daily but one p_H reading made on a sample of secretion. A study of this type, while informative, has distinct limitations, since the nasal secretions are not in situ, but are divorced from the normal milieu of the nose and are susceptible to changes in p_H on exposure to air.

Some of the problems encountered are illustrated by Hilding,[1] who employed a series of standardized indicators to test the p_H values of a number of samples of nasal secretion collected in the course of the common head cold. The results indicated a definitely alkaline reaction, varying between 7.2 and 8.3. In order to determine the extent and speed of the changes in p_H, single samples from a patient with a cold were tested during the first fifteen to thirty minutes. There was a change from 7 to 8.5 during a few minutes. Thereafter there seemed to be little or no change. Hilding found that the rapid change in p_H during the first fifteen minutes was due to the loss of carbon dioxide gas that had been absorbed from the expired air as it passed through the nose.

In order to eliminate some of the disadvantages inherent in the methods employed elsewhere and to analyze further the wide range of p_H findings reported, I decided to study the p_H of nasal secretions in situ by utilizing a silver–silver chloride glass electrode designed for the nose in conjunction with the Coleman electrometer. By conducting minute to minute readings of the p_H values of nasal secretions in situ in a group of patients observed daily for more than an entire year and graphically charting the results, it is possible to explain satisfactorily the wide range of p_H determinations revealed elsewhere by workers employing other methods of proceduce in examining the secretions of the nose under clinically normal conditions and under the conditions of acute rhinitis, acute rhinosinusitis and allergic rhinitis.

METHOD

The Coleman electrometer is used in the determination of nasal p_H. The calomel half-cell is utilized as a reference electrode, contact with the patient being made by means of a salt bridge. The salt bridge contains a saturated solution of potassium chloride, which is separated from a physiologic solution of sodium chloride by a plug of agar prepared in a concentrated solution of potassium chloride. The reference cell is submerged in the saturated solution of potassium chloride. The

1. Hilding, A.: Ann. Otol., Rhin. & Laryng. **43**:47, 1934.

apparatus is standardized by submerging both the agar end of the salt bridge and the glass electrode in a small beaker filled with Sorensen's buffer solution.

The glass electrode is inserted into the patient's naris and is left resting on the floor of the nasal passageway in contact with the inferior turbinate. The tube containing the salt bridge makes contact with the patient by being placed on the opposite cheek, immediately lateral to the opposing naris. The salt bridge is placed on the side opposite the glass electrode.

It is advisable that both the glass electrode and the salt bridge rest on a specially constructed pillow lying on the patient's chest. This will eliminate any unnecessary shifting of the electrode and the salt bridge, a consideration that is

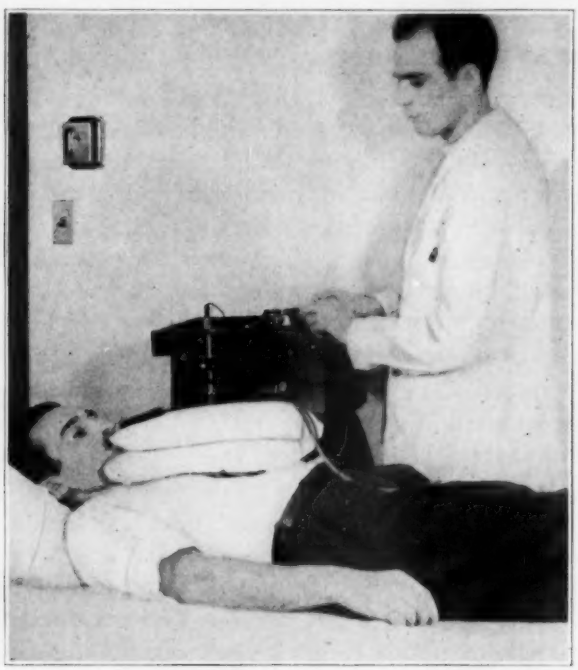

Fig. 1.—The position of the patient in determining the p_H of nasal secretion in situ.

important, for movement will distort the accuracy of the readings. The patient should remain as quiet as possible. Readings are taken each minute (fig. 1).

The calomel reference electrode was checked both at the beginning and at the end of each experiment, and no appreciable change in its potential was observed. Before each experiment the standard cell was checked against a standard buffer solution. At monthly intervals the buffer solution was standardized against a number of precision hydrogen electrodes. A Coleman electrometer of the type employed has a standard error of $\pm .01$ p_H. The differences in potential induced by the differences in temperature of the nasal mucosa as compared with the room temperature were compensated for by a temperature adjustment on the electrometer.

The p_H of nasal secretion in situ describes a series of high and low points (fig. 2). At no time does it remain constant over an appreciable period. I find that the fluctuations of nasal p_H in a clinically normal nose range from approximately 5.5 to 6.5. The futility of relying on only one p_H reading is amply illustrated by the frequent numerical changes in p_H values that occur when readings are recorded over one hour or more. Particularly do I wish to emphasize this phenomenon, since it would be a mistake to standardize—to press all observations into definite and fixed levels and apply these levels to patients, quite overlooking the fact that each chemical level can be considered only in relation to one person at a particular time and under certain conditions and none other. In so doing one may miss the variation that is so obvious when minor changes are followed in the nasal p_H from day to day, from hour to hour, yes, from minute to minute!

Petersen [2] has demonstrated that in the p_H level there is presented one of the major balances with which every organic and every inorganic

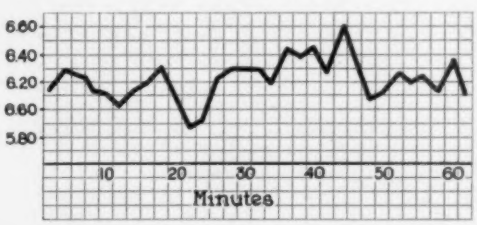

Fig. 2.—A graph of the p_H of nasal secretion in situ. Minute to minute recordings were taken for one hour.

reaction is integrated. For the time being, I shall merely call attention to the fact that the nasal p_H varies with sleep, with rest, with the ingestion of food and diurnally and nocturnally and can be changed not only by infection but by alterations in a person's emotions. The significance of fluctuations in p_H must be understood, because with such changeability alterations in the permeability of membranes and in the stimulation of tissues— indeed every phenomenon of pathologically altered tissue— will naturally be reflected in the milieu of the cells. The nasal p_H can be said to mirror a whole series of functional as well as chemical changes which may be of clinical significance in the inadequate organ or for the organism as a whole. An understanding of the peaks and intervening troughs of the charts may perhaps contribute to an understanding of the beginning of nasal disease processes.

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2. Petersen, W. F.: The Patient and the Weather, Ann Arbor, Mich., Edwards Brothers, Inc., 1936, vol. 1, pt. 2.

The series of high points and low points which nasal p_H describes is a biologic phenomenon that is equally characteristic of mucous membrane surfaces about the head and neck and of the skin. Studies were made of the p_H of the buccal mucosa, the lips and various portions of the tongue (fig. 3) and of the gingiva at intervals of one and two minutes. The same variation which is observed in the p_H of nasal mucosa is also observed in the p_H of the buccal mucosa, the lips, the tongue, the gingiva and the skin. It is reasonable to assume that a similar variation may be found for the p_H of vaginal mucosa or of rectal mucosa—in fact, for that of any surface of the body that readily lends itself to study in situ by means of the glass electrode. It is quite conceivable that this method of study when applied to these regions may bear definite therapeutic fruit.

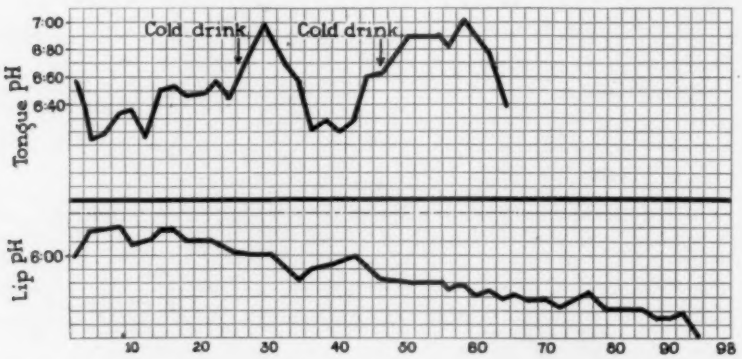

Fig. 3.—The p_H of the secretions on the tongue and the lip. A drift toward alkalinity followed the taking of a drink of cold water.

In one of my earliest experiments, I attempted to correlate the p_H of the nasal mucosa and that of the buccal mucosa by charting the p_H values obtained during alternate minute to minute readings. In a general way, it was found that the fundamental changes in p_H of the secretions on the nasal and buccal mucosa progressed in a parallel manner and that this parallelism was disturbed only by the application of an ice bag to the patient's head (fig. 4). Similarly, in measuring the p_H of the tip of the tongue and that of the buccal mucosa it was found that the p_H fluctuations of these two surfaces ran parallel. However, shortly after the patient drank ½ glassful of water, the parallelism was broken (fig. 5). Since in the act of drinking cold water a greater surface contact is produced between the liquid and the tongue than between the liquid and the buccal mucosa, the effect is a more pronounced trend toward alkalinity at the surface of the tongue than in the buccal mucosa. The

undulating p_H variation of the skin, too, is affected by friction and by the application of ice to the skin (fig. 6).

The course of nasal p_H variation is altered by the application of either cold or heat. An ice bag applied to each side of the nose for twenty minutes produced a drift toward alkalinity. When the ice bag was removed, the nasal p_H drifted toward a more acid status. The effect of heat was readily demonstrated by a characteristic response—diametrically

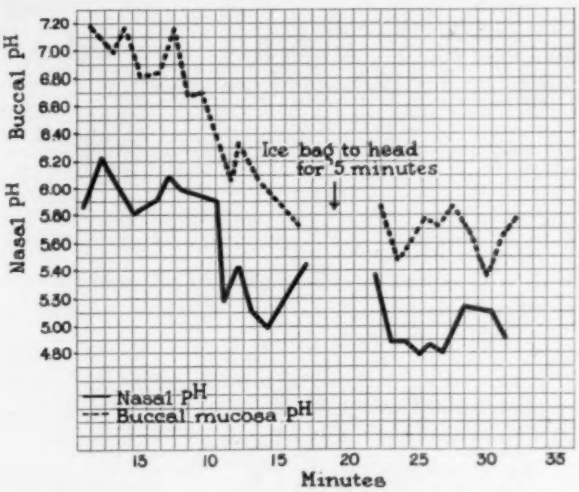

Fig. 4.—The effect of the application of an ice bag to a patient's head on the course of the p_H of the secretions of the nose and the buccal mucosa.

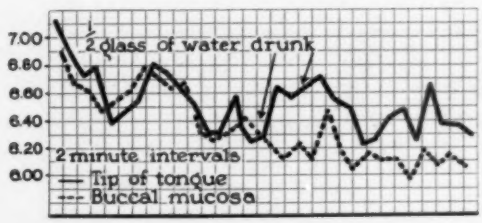

Fig. 5.—The effect of cold water on the p_H of the secretion on the tip of the tongue and on the buccal mucosa.

opposed to that produced by cold. An electric heater lamp placed at a distance of 2 feet (61 cm.) from the patient's face was permitted to glow for fifteen minutes. At the end of this period, the nasal p_H proceeded to drop, i. e., nasal secretions became more acid (fig. 7). During this experiment the electrodes were removed while the heat was applied and for fifteen minutes subsequent to this, in order to avoid the influence of

the increasing temperature on the electrodes. Again, cold water sprayed into the nasal passage produced a drift toward alkalinity, while warm water provoked a drift toward acidity. These characteristic responses of the course of nasal p_H to cold and heat could be repeated again and again.

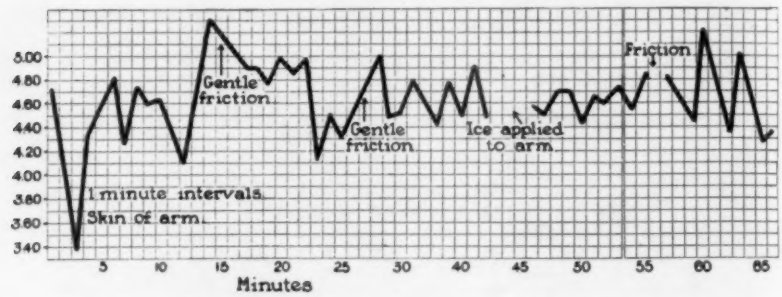

Fig. 6.—The effect of friction and the application of ice to the skin on the p_H of the cutaneous secretions.

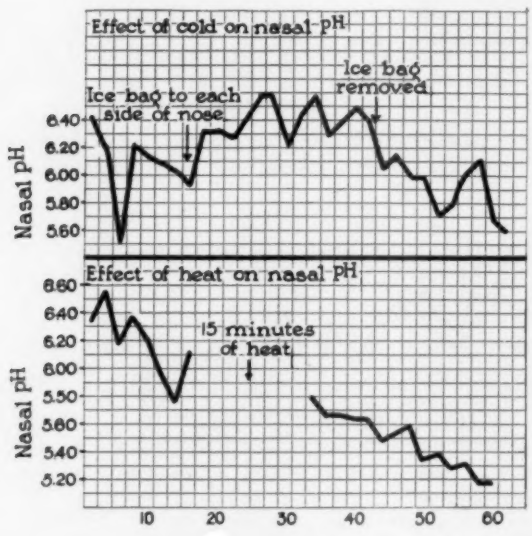

Fig. 7.—The effect of cold and heat on nasal p_H.

A series of experiments was then conducted to study the effect of a number of nasal vasoconstrictors manufactured by various pharmaceutic houses throughout this country. Before beginning these experiments I was convinced that all of the vasoconstrictors would alter the variation

of the nasal p_H in a like manner. To my great surprise they did not. The following list shows the nasal vasoconstrictors utilized, together with their p_H values as observed.

Preparations Containing in Part:	p_H
Ephedrine	
(a) Metaphedrine aqueous	9.5
(b) Metaphedrine isotonic	9.5
(c) Isohalant	6.0
(d) Gluco-fedrin	5.4 to 5.8
Ephedrine hydrochloride	
(a) Ephedrine hydrochloride 1 per cent in a physiologic solution of sodium chloride	5.9
Epinephrine (hydrochloride)	
(a) Adrenalin inhalant 1:1,000	2.3 to 2.5
(b) Synthetic epinephrine 1:1,000	3.7
Propadrine hydrochloride	
(a) Propadrine hydrochloride 1 per cent	4.8 to 5.0
Neo-synephrin hydrochloride	
(a) Neo-synephrin hydrochloride 0.25 per cent	3.5 to 4.2
(b) Neo-synephrin hydrochloride 1 per cent	4.1 to 4.2
Amphetamine	
(a) Benzedrine solution (when applied to the nose)	8.0

It will be observed that the p_H of the nasal vasoconstrictor varies markedly from 2.3 (acid) to 9.5 (alkaline). It soon became evident to me that the manner in which the trend of the nasal p_H variation is affected depends entirely on the p_H of the drug employed. Thus, drugs with a highly alkaline hydrogen ion concentration (such as metaphedrine aqueous, metaphedrine isotonic [fig. 8] and amphetamine [benzedrine] solution) induce in the nasal p_H variation a marked drift toward alkalinity. Drugs with a p_H range of 5.5 to 6.5 (such as 1 per cent solution of ephedrine hydrochloride in saline solution, gluco-fedrin and isohalant), which approximate the normal fluctuations of nasal p_H do not markedly affect the nasal p_H one way or another when the study is carried out for one hour and when the nasal p_H for that particular day swings within the normal range (fig. 9). Drugs with a p_H level of less than 5 induce a noticeable drift downward in the nasal p_H, i. e., toward acidity. Within this group lie both preparations of neosynephrine hydrochloride, synthetic epinephrine 1:1,000, adrenalin inhalant (a preparation of epinephrine hydrochloride) 1:1,000 and, to a lesser extent, propadrine hydrochloride 1 per cent (fig. 10).

On many occasions the drift in the nasal p_H was prolonged for an hour, and frequently longer; thus sufficient time elapsed for alterations

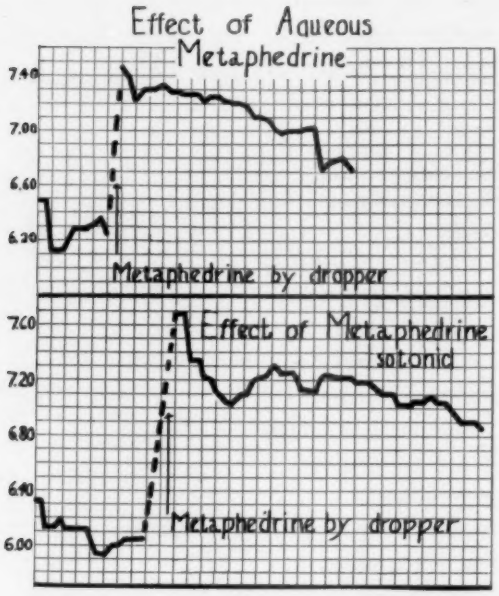

Fig. 8.—The drift toward alkalinity in the fluctuations of the nasal p_H induced by metaphedrine aqueous and metaphedrine isotonic solutions.

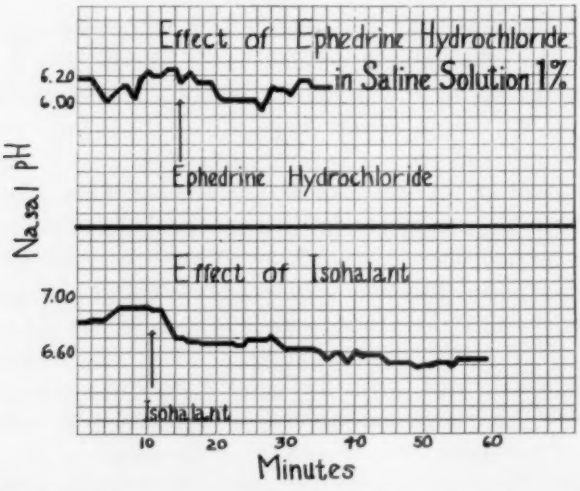

Fig. 9.—The relatively even tenor in nasal p_H after the use of 1 per cent ephedrine hydrochloride in a solution of sodium chloride and by isohalant (a preparation of ephedrine). Similar responses followed the administration into the nose of 3 per cent ephedrine hydrochloride and gluco-fedrin (a preparation of ephedrine).

to occur in the permeability of the nasal capillaries and in that of the mucous membrane. On other occasions the nasal vasoconstrictors altered the drift in nasal p_H for shorter periods. This phenomenon affords many interesting problems, for unquestionably in such instances not only must the ever changing role of the host be considered but also the effect of temperature and barometric pressure on the action of drugs. That

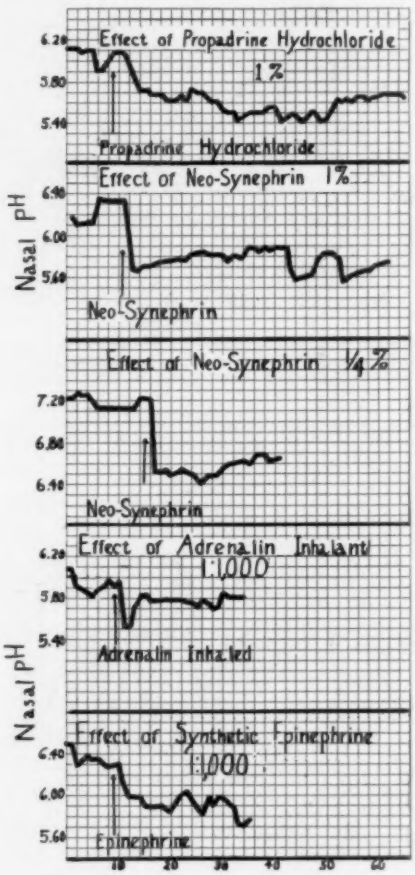

Fig. 10.—The drift toward acidity in nasal p_H produced by neo-synephrin hydrochloride, 1 per cent and 0.25 per cent; propadrine hydrochloride solution 1 per cent, adrenalin inhalant 1:1,000 and synthetic epinephrine 1:1,000.

drugs act differently under changes in barometric pressure and temperature has been shown by Macht [3] in his experiments on digitalis and by Nedzel [4] in his work on morphine.

3. Macht, D. I., in discussion on Fenn, G. K., and Gilbert, N. C.: Anginal Pain as a Result of Digitalis Administration, J. A. M. A. **98**:99 (Jan. 9) 1932.

4. Nedzel, A. J.: J. Lab. & Clin. Med. **23**:1063, 1938.

The use of an amphetamine (benzedrine) inhaler produced a marked rise toward an alkaline status when two inhalations were made by the patients. The change in nasal p_H became dramatically evident within one or two minutes (fig. 11). When the amphetamine was inhaled through one naris, the p_H of the mucosal secretion was affected only in

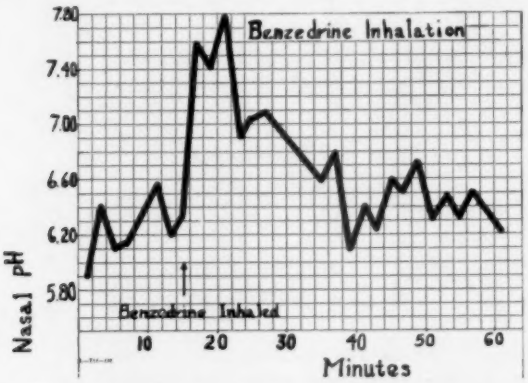

Fig. 11.—The alkaline response in nasal p_H to inhalations from an amphetamine (benzedrine) inhaler.

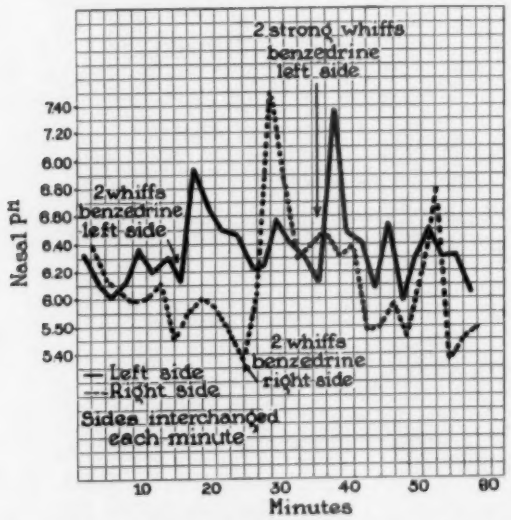

Fig. 12.—The effect of amphetamine (benzedrine) inhalation on nasal p_H when the inhaler was applied alternately to the nasal passageways.

that particular naris, whereas the secretion in the unaffected nasal passageway showed no change in p_H. But when the unaffected naris was subjected to an inhalation from the inhaler, a marked drift toward an alkaline status was soon discovered (fig. 12). To establish further the effect of alkaline and acid solutions on the course of nasal p_H, two solu-

tions were used—Sorensen's solution with a p_H of 8.36 and dilute acetic acid with a p_H of 3.35. When sprayed in the nose, the dilute acetic acid attracted the nasal p_H toward a definitely acid status, while the alkaline Sorensen's solution drew the nasal p_H toward a markedly characteristic alkaline status.

Hilding,[5] collecting samples of nasal secretions, observed that the p_H rose slightly and became more alkaline during the first four days of acute rhinitis. Mittermaier [6] found that in cases of acute inflammation the p_H of the nasal secretions he collected was definitely on the acid side. The results of my own studies of the p_H of nasal secretions in situ during an attack of acute rhinitis parallel the findings of Hilding. For the most part, I find that the p_H of nasal secretions in situ during an attack of acute rhinitis or of acute rhinosinusitis is alkaline—the secretions are more alkaline than normal. This finding has clinical significance, for those nasal vasoconstrictors which can lower the nasal p_H from an abnormal alkaline status to a desirably normal acid status may perform a valuable function. Conversely, those vasoconstrictors which raise, enhance or perpetuate an alkaline nasal p_H during acute rhinitis may prolong an undesirable status of nasal p_H—a status in which the bacteria accompanying the acute infection find a fertile field for growth. It is more than likely that during alkalosis of the nasal mucosa, changes in capillary, arterial and membrane permeability proceed at so significant a pace as to prolong an abnormal physiologic nasal environment.

At present, work is being done to show that the numbers of bacteria that can be recovered from the nasal mucosa at high and low points in the variation of the nasal p_H differ astonishingly. The evidence for this will be presented at some future time. To proceed—vasoconstrictors possessing an unusually high p_H value may merely serve to disrupt the normal p_H value of the nasal secretion found in the clinically normal nose, enhance irritation of the nasal mucosa and postpone the return to a normal p_H status. It is therefore suggested that during an attack of acute rhinitis or acute rhinosinusitis the employment of a nasal vasoconstrictor which lowers the alkaline p_H to a level between 5.5 and 6.5—approximating the normal p_H status of nasal secretions in situ—is most desirable. This, of course, implies that there be a standardization of the p_H values of the various nasal vasoconstrictors now available to the public. Contrary to the generally accepted American advertising slogan, it is now time to acidify the nasal mucosa—not to alkalinize!

A study of the p_H of nasal secretions in cases of nasal allergy suggests a similar state of affairs. Buhrmester [7] found that nasal secretions collected from patients with nasal allergy were for the most part predomi-

5. Hilding, A.: The Common Cold, Arch. Otolaryng. **12**:133 (Aug.) 1930.
6. Mittermaier, R.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **127**:1, 1930.
7. Buhrmester, C. C.: Ann. Otol., Rhin. & Laryng. **42**:1041, 1933.

nantly alkaline, but instances were noted in which the p_H remained acid. These findings were substantially supported by my own studies of nasal secretions in situ. An alkaline status of the nasal secretions was seen during the active stages of allergic rhinitis. The nasal p_H descended to the neutral point and to the acid status when clinical improvement was felt by the patient. Whenever the symptoms became aggravated, a return to alkalosis was detected. Therapeutically the implications for the desirable p_H of nasal vascoconstrictors becomes obvious. As in the case of the acute rhinitis, I believe that the use of an appropriate nasal vascoconstrictor implies the selection of one that will lower the alkaline p_H to a normal acid level, between 5.5 and 6.5.

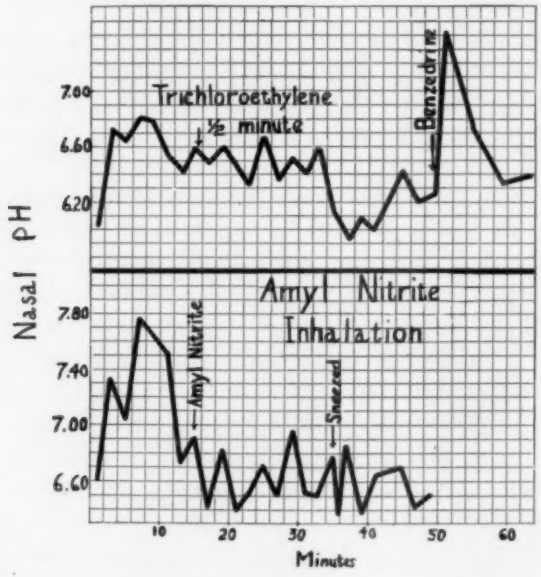

Fig. 13.—The slow drift toward acidity in nasal p_H induced by the inhalation of amyl nitrite and trichloroethylene. The characteristic rise to alkalosis occurs after an inhalation of amphetamine.

Finally a brief investigation was made of the effect of vasodilators on the nasal p_H. Since I was unable to obtain a satisfactory nasal vasodilator, I employed two drugs that are frequently inhaled during attacks of angina pectoris. These are amyl nitrite and trichloroethylene. The use of these drugs induced in the nasal p_H a slow drift toward acidity (fig. 13).

SUMMARY AND CONCLUSIONS

With the advent of comparatively new and simple methods for measuring hydrogen ion concentration, it is possible to awaken the clinician's interest in a type of investigation that has practical application to

innumerable and diversified fields. To offset the fact that nasal secretions, when collected, are susceptible to changes in p_H almost instantly when they are exposed to air and are divorced from the normal milieu of the nose, the use of the glass electrode with its associated measuring equipment, the Coleman electrometer, has made it feasible to measure the p_H of nasal secretions in situ with ease and precision.

By conducting minute to minute readings of the p_H values of nasal secretions in situ in a group of patients observed daily for more than a year and graphically charting the results, it has been possible to explain adequately the wide range of nasal p_H revealed in contributions elsewhere by workers employing other methods of procedure in studies of clinically normal nasal conditions and of acute rhinitis, acute rhinosinusitis and allergic rhinitis.

The p_H of nasal secretions in situ describes a series of high and low points. At no time does the p_H remain constant over an appreciable period. Its fluctuations in a clinically normal nose—from approximately 5.5 to 6.5—appear to follow a definite course. The futility of relying on only one p_H reading is amply illustrated by the frequent numerical changes in p_H values that occur when readings are recorded over one hour or more.

The course of nasal p_H is altered by the application of either cold or heat. Cold produces a drift toward alkalinity, heat a drift toward acidity. The p_H of nasal secretions in situ during an attack of acute rhinitis or acute rhinosinusitis or during the more active phases of an attack of allergic rhinitis is alkaline. This finding has clinical significance, for those nasal vasoconstrictors which can lower the nasal p_H from an abnormal alkaline status to a normal acid status—a p_H level between approximately 5.5 and 6.5—may perform a valuable function. Conversely, those nasal vascoconstrictors which raise, enhance or perpetuate an alkaline nasal p_H during these rhinologic conditions may prolong an undesirable status of nasal p_H.

It is suggested that during an attack of acute rhinitis or acute rhinosinusitis and the more active stages of an attack of allergic rhinitis the employment of a nasal vascoconstrictor, such as 1 per cent ephedrine hydrochloride in saline solution, which lowers the alkaline p_H to an acid level between 5.5 and 6.5 is most desirable. It is suggested further that there be a standardization of the p_H values of the various nasal vasoconstrictors at present available to the public. Contrary to the generally accepted American advertising slogan, it is now time to acidify the nasal mucosa—not to alkalinize!

185 North Wabash Avenue.

Case Reports

SINUSITIS AND STAPHYLOCOCCUS AUREUS BACTEREMIA; RECOVERY

HENRY H. BEINFIELD, M.D., BROOKLYN

Staphylococcus aureus infection of the blood stream has always been considered with grave concern by all physicians because of the lack of specific therapeutic measures to cope with it properly and check its progress. Chemotherapy is now playing an increasingly important role in the treatment and control of acute infections of all types. Much has been said for the use of sulfanilamide and sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) in this connection, but little has been reported on the use of these drugs and the results obtained in the treatment of Staph. aureus infections.

The present interest in the treatment of staphylococcic bacteremia has prompted this report for its academic value.

REPORT OF A CASE

F. W., a boy 8 years of age, was first seen by me on Aug. 31, 1940. He had first become sick on August 29, complaining of severe frontal headache, general malaise, running nose, redness of both eyes and a slight fever. On August 30 his temperature reached 101.5 F., the left eyelids became markedly swollen, the left nostril became obstructed and in general he felt very sick. He had never complained of a postnasal drip or any other trouble with his nose. There was no history of injury. Although he indulged a great deal in swimming and diving during the summer, he had not been in the water for three weeks before this illness. He had not had a cold for eight months. Examination revealed a moderate swelling on the left side of the forehead, extending from the left eyelids, which were tensely chemotic and slightly ecchymotic. The left eyeball was congested but freely movable. There was moderate tenderness in the region over the left frontal sinus and around the swelling on the forehead. The entire nasal mucous membrane was so congested and edematous that it completely obstructed the left naris. Over the left inferior turbinate was a fibrinous exudate, which did not leave a bleeding surface on removal. The right naris was clear. Twelve grains (0.78 Gm.) each of sulfanilamide and sodium bicarbonate were given every four hours (the patient weighed 72 pounds [32.7 Kg.]). Three per cent ephedrine administered in the form of nasal drops and nasal packs failed to produce any shrinkage of the nasal mucous membrane. Inhalations of steam were used with little or no effect. Cold compresses saturated with a 50 per cent solution of magnesium sulfate were applied constantly to the swelling on the forehead.

On September 1 all of the symptoms became aggravated, and the temperature rose to 104 F. The patient did not have chills at any time during his entire illness. He was now admitted to the Prospect Heights Hospital. Physical examination gave negative results. The spleen was not enlarged, the neck was not rigid and the Kernig and Babinski signs were absent. Fluctuation was elicited over the swelling on the left side of the forehead; this was aspirated with a heavy needle on a Luer syringe, and 1 cc. of a thick reddish fluid was obtained and cultured. A blood culture and a culture of material from the left nostril were also

made. The edema spread from the left side to the right upper eyelid, partially closing the right eye. Roentgen examination of the sinuses showed a veiling of the region of the left antrum suggestive of displacement of the normal air content with fluid. The frontal sinuses were immaturely developed and very shallow but apparently pneumatic. The sphenoid sinus was large and well filled with air, and the ethmoid sinuses were poorly developed but obviously air filled. The red blood cells numbered 3,900,000 and the white blood cells 16,500 per cubic millimeter, with polymorphonuclears 72 per cent.

On September 3 the report on the cultures of material taken from the left nostril, pus aspirated from the forehead and the blood showed the presence of

Graphic representation of the patient's condition from admission to discharge.

Staph. aureus, with twenty-five colonies per cubic centimeter in the blood. The administration of sulfanilamide was now discontinued, and another blood culture was made. Twelve grains (0.78 Gm.) of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) was then given every four hours. The hemoglobin content of the blood was 80 per cent. The white blood cells numbered 24,500 per cubic millimeter, with polymorphonuclears 81 per cent. The urine showed a neutral reaction, with albumin (2 plus), occasional white blood cells and no casts. The temperature was sustained between 102.6 and 104.4 F. until September 4, when it dropped to 101.4 F. The swelling of the left eye and of the forehead, however, increased, with no change in the swelling of the right eye. The swelling over the forehead was now incised and drained. About 10 cc. of a reddish gray fluid

was evacuated, exposing bare bone. This bone was probed and found to be smooth. To avoid any unnecessary trauma, no effort was made to search for a perforation or for the source of the pus. The wound was left open and drained with rubber tissue. The left antrum was punctured underneath the inferior turbinate with a Douglas antrum trocar, and suction was employed instead of irrigation. The blood culture made on September 3, before the administration of sulfathiazole, showed a reduction of Staph. aureus to two colonies per cubic centimeter. From this point on the patient began to improve steadily, with all signs and symptoms gradually disappearing. The value for hemoglobin was 72 per cent. The white blood cells numbered 28,950 per cubic millimeter, with polymorphonuclears 83 per cent. The administration of sulfathiazole was discontinued on September 8, after the temperature returned to normal.

A third blood culture, made on September 9, was negative. The value for hemoglobin was 65 per cent. The white blood cells numbered 22,050, with polymorphonuclears 68 per cent.

On September 10 a pustule was found on the scalp over the left temporal region, from which a grayish yellow pus was expressed. Examination of the blood on September 11 showed the hemoglobin content 76 per cent, white blood cells 11,400 and polymorphonuclears 75 per cent. On September 12 a roentgenogram of the sinuses showed a continued veiling of the region of the left antrum, and there was also now some veiling of the left ethmoid field. There was no visible evidence of change in the detail of the frontal field.

The patient was discharged from the hospital September 14.

COMMENT

There was never any chill or septic temperature to lead one to suspect the presence of bacteremia.

The first blood culture was made primarily with the thought of the possibility of a thrombosis of the cavernous sinus and also because of the rapid development of symptoms accompanied by a sudden rise in temperature.

The blood culture was positive two days after the onset of illness. Sulfanilamide was given more or less routinely on the presumption that a streptococcic infection was present. When the report of the first blood culture showed twenty-five colonies of Staph. aureus per cubic centimeter, sulfathiazole was immediately substituted. This was done in the belief that the result of the second blood culture, made before the administration of sulfathiazole, would be the same as or worse than the first, with the possibility also that the condition was unaffected by the sulfanilamide. Instead, the second blood culture showed the number of colonies to be reduced to two per cubic centimeter, which signified that the number of colonies was reduced while the patient was taking sulfanilamide.

The subperiosteal abscess was incised on September 4, after the patient had taken sulfathiazole for twenty-four hours. This waiting period was selected with the idea that there would be a sufficient amount of sulfathiazole in the circulation to prevent any untoward results that might follow surgical intervention.

Since the result of the second blood culture was not reported until September 5 and since the patient was improving, the administration of sulfathiazole was continued until the temperature became normal.

My purpose in using suction to drain the left antrum was primarily to avoid the trauma which might follow the employment of any degree of pressure needed to wash the contents from the antrum.

Acute sinusitis involving the ethmoid sinuses, the frontal sinuses and the antrums is frequently complicated in children by osteomyelitis of the calvarium, terminating in sepsis and meningitis. The literature is replete with reports of such cases (Sobol [1]; Yaskin [2]; Sjöberg [3]). The conservative measures employed in the case reported here are in accord with those advocated by Goodyear [4] and by Simpson,[5] who are of the opinion that radical surgical procedure should be postponed as long as possible, to permit localization and walling off and the external drainage of a definite collection of pus without further intervention. Mellon, Shinn and McBroom [6] found by experimental and clinical experience that staphylococcic infections are either uninfluenced or at best slightly affected by sulfanilamide. Their observations are not borne out by the facts in the case reported here, because the sulfanilamide reduced the number of colonies of Staph. aureus in the blood from twenty-five to two per cubic centimeter in three days.

According to Barlow,[7] sulfathiazole prolonged the life of mice infected experimentally with a strain of Staph. aureus virulent for mice and prevented the development of or allowed the healing of abscesses in the kidneys and other organs of a significant number of animals. Carroll [8] and others reported the use of sulfamethylthiazole (2-sulfanilamido-4-methylthiazole) in 2 cases of staphylococcic septicemia in which there was a positive blood culture and in which the condition did not respond to treatment with sulfanilamide or any of its other derivatives.

Fitch [9] described a case in which fifty colonies of Staph. aureus in the blood cleared under treatment with sulfathiazole. Blood cultures made daily showed a gradual reduction in the number of the colonies in the

1. Sobol, I. M.: Clinical Observations on the Role of Sinuses in Complications of Orbital Inflammation, Vestnik oftal. **13**:451, 1938.

2. Yaskin, J. C.: Neurologic Complications of Infections of the Temporal Bone and Paranasal Sinuses, Arch. Otolaryng. **30**:157 (Aug.); 360 (Sept.) 1939.

3. Sjöberg, A. A.: A Contribution on the Prognosis and Treatment of the Rhinogenous and Otogenous Brain Abscess, Acta oto-laryng. **27**:638, 1939.

4. Goodyear, H. M.: The Indication for Surgery in Meningitis Secondary to Disease of the Middle Ear and of the Nasal Sinuses: Reservations in Early Surgical Intervention, Laryngoscope **49**:102 (Feb.) 1939.

5. Simpson, W. L.: Surgical Treatment and Its Complications in Cases of Acute Sinusitis, Arch. Otolaryng. **32**:250 (Aug.) 1940.

6. Mellon, R. R.; Shinn, L. E., and McBroom, J.: Therapy of Experimental Staphylococcus Infections with Sulfonamide Compounds, Proc. Soc. Exper. Biol. & Med. **37**:563, 1937.

7. Barlow, O. W., and Homberger, E.: Specific Chemotherapy of Experimental Infections with Thiazole Derivatives of Sulfanilamide, Proc. Soc. Exper. Biol. & Med. **42**:792 (Dec.) 1939.

8. Carroll, G., and others: Sulfamethylthiazole: A Report of Its Clinical Use in Staphylococcus Septicemia with Apparent Success; Report of Animal Experiments, South. M. J. **33**:83 (Jan.) 1940.

9. Fitch, T. S. P.: Sulfathiazole in Epidermal Abscess with Septicemia and Pyaemia, Arch. Pediat. **57**:119 (Feb.) 1940.

blood stream. Five transfusions were given in conjunction with the administration of sulfathiazole, and recovery followed.

Herrell and Brown [10] employed sulfamethylthiazole in combating Staph. aureus infections in vitro and in vivo and found this drug to be more specific than sulfathiazole. They also pointed out that sulfanilamide is slightly less active than sulfathiazole.

Harrison [11] cited a case similar to the one reported here, that of a boy of 16 who was given azosulfamide [12] after a frontoethmoid operation and who recovered three weeks later. No mention, however, was made of cultures or roentgenograms having been made.

Goldberg and Sachs [13] used sulfapyridine in the treatment of bacteremia due to Staph. aureus and cited 2 cases in which the blood became sterile after the use of the drug.

Pegan and Reed [14] reviewed a case of staphylococcic septicemia in which the patient recovered after having been given seven transfusions by immunized donors. This treatment was supplemented by the administration of sulfapyridine. A negative blood culture was obtained on the twelfth day. The authors expressed the belief that sulfapyridine has a selective affinity for Staphylococcus.

Uren [15] stated that necrosis of the roof of the maxillary sinus and the external wall of the ethmoid may extend through the periorbital tissue and produce orbital cellulitis or an orbital phlegmon. Transmission of inflammatory processes from the paranasal sinuses to the orbit by way of the venous anastomoses occurs perhaps the most frequently. In my case I was content to evacuate the subperiosteal abscess on the forehead with the least amount of trauma, and I did not deem it advisable to explore this area in an effort to locate a perforation or the source of the pus. It is questionable whether the pus came from a perforation in the ethmoid area or followed a cellulitis or resulted from a retrograde thrombosis of the veins leading from the maxillary sinus.

SUMMARY

A case of bacteremia due to Staph. aureus is reported.

Cultures of material taken from the nose and of blood and pus all showed Staph. aureus.

A pustule developed on the skin.

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10. Herrell, W. E., and Brown, A. E.: The Clinical Use of Sulfamethylthiazole in Infections Caused by Staphylococcus Aureus: Preliminary Report, Proc. Staff Meet., Mayo Clin. **14**:753 (Nov. 29) 1939.

11. Harrison, H. H.: A Case of Osteomyelitis of the Frontal Bone Complicating Left-Sided Frontal Sinusitis, Australian & New Zealand J. Surg. **8**:313 (Jan.) 1939.

12. Azosulfamide is disodium 4-sulfamidophenyl-2′-azo-7′acetylamin-1′-hydroxynaphthalene-3′, 6′-disulfonic acid. This substance has been known as prontosil soluble, as prontosil and as neoprontosil.

13. Goldberg, S. L., and Sachs, A.: Sulfapyridine in the Treatment of Staphylococcus Aureus Bacteremia, J. A. M. A. **113**:1639 (Oct. 28) 1939.

14. Pegan, P. M., and Reed, C. C.: Case of Staphylococcus Septicemia Which Recovered by the Use of Sulfapyridine, J. M. Soc. New Jersey **37**:111 (March) 1940.

15. Uren, C. T.: Serious Complications of Frontal Disease, Ann. Otol., Rhin. & Laryng. **48**:515 (June) 1939.

Sulfanilamide reduced the number of colonies of Staph. aureus in the blood from twenty-five to two per cubic centimeter in three days.

The first report of Staph. aureus in the blood stream prompted the use of sulfathiazole because the result of the second blood culture, made before the administration of sulfathiazole, was not known at that time.

CONCLUSION

Sulfanilamide rid the blood stream of an infection due to Staph. aureus.

The action of sulfathiazole cannot be properly evaluated in this case, because of the previous use of sulfanilamide.

Roentgenograms were taken by Dr. Frederick E. Elliott, roentgenologist, of the Prospect Heights Hospital.

760 Eastern Parkway.

CONGENITAL CYST OF THE LARYNX

S. F. Crynes, M.D., and S. C. Yinger, M.D., Springfield, Ohio

Laryngeal cysts are not as rare as recent textbooks and periodicals would indicate (Myerson [1]), although they are probably far less frequent than reported in earlier periods, when many laryngeal polyps were mistaken for cysts. Of the benign cysts of the larynx, those of congenital origin are by far the most uncommon. We are therefore prompted to report a case of laryngeal cyst of this type which recently came under our observation.

REPORT OF A CASE

A 47 year old white woman, while eating some coarse food, suddenly noticed pain on swallowing, which she referred to the left side of the neck. The patient was seen by one of us (S. C. Y.) when it was supposed that these symptoms were due to the lodgment of a foreign body. There was no preceding history of infection of the upper respiratory tract or of painful or difficult swallowing.

Examination of the patient revealed slight tenderness to pressure in the region of the posterior part of the thyroid cartilage on the left. Mirror examination revealed no foreign body in the pharynx, larynx or piriform sinus. In the left aryepiglottic fold there was a small rounded elevated mass, covered by intact reddened mucous membrane. The remainder of the larynx was entirely normal in appearance. The rounded mass, which could be palpated with the end of a probe, was soft, fluctuant and compressible and was recognized as a cyst. Since the patient's symptoms persisted, the cyst was removed three days later under direct laryngoscopic inspection by the use of an angular cupped forceps, with the region under local anesthesia. After removal of the cyst, a small crater was left in the aryepiglottic fold. The patient left the hospital two days later and has been observed several times since. At the time of this report (thirteen months after removal) the larynx is normal and there is no evidence of recurrence of the cyst.

Pathologic Report.—The cyst was ruptured during the operation. It measured 7 mm. in diameter and contained a clear thin fluid. A small fragment of tissue, consisting of a white membrane measuring 0.2 by 0.3 by 0.15 cm., was submitted for histologic examination. The histologic section showed two rather thick layers of stratified squamous epithelium covering a narrow zone of mesenchymal tissue. One of the epithelial layers represented the lining of the laryngeal mucosa, while the other formed the epithelial lining of the cyst. The layers were about equal in thickness and showed a distinct and regular basic membrane and good keratinization of the superficial epithelial layers. In the inner epithelial layer a few mitoses could be seen. Beneath the epithelium a thin layer of hyaline connective tissue fibers was noted, in which small capillaries and scattered lymphocytes could be observed. The largest portion of the specimen removed was composed

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From the Springfield City Hospital and the Department of Pathology, Ohio State University.

1. Myerson, M. C.: Cysts of the Larynx, Arch. Otolaryng. **18**:281-290 (Sept.) 1933.

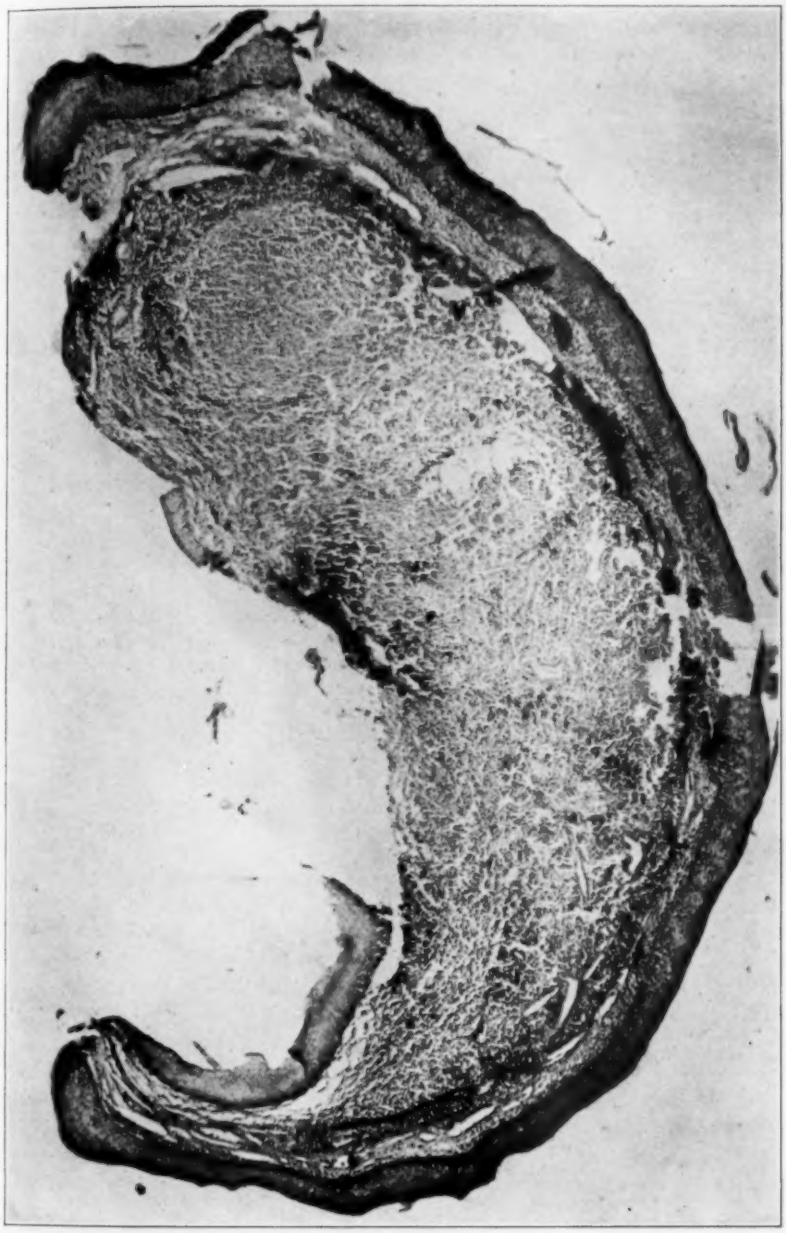

Fig. 1.—Section through the entire thickness of the cyst wall (× 25). At the upper left is a large lymph follicle. The lumen, being of squamous epithelium, was broken in sectioning.

of lymphoid tissue, which showed a follicular arrangement and contained large germinal centers. No glandular structures of any kind could be observed. There was no cartilage or bone. The pathologic diagnosis was laryngeal cyst lined with squamous epithelium.

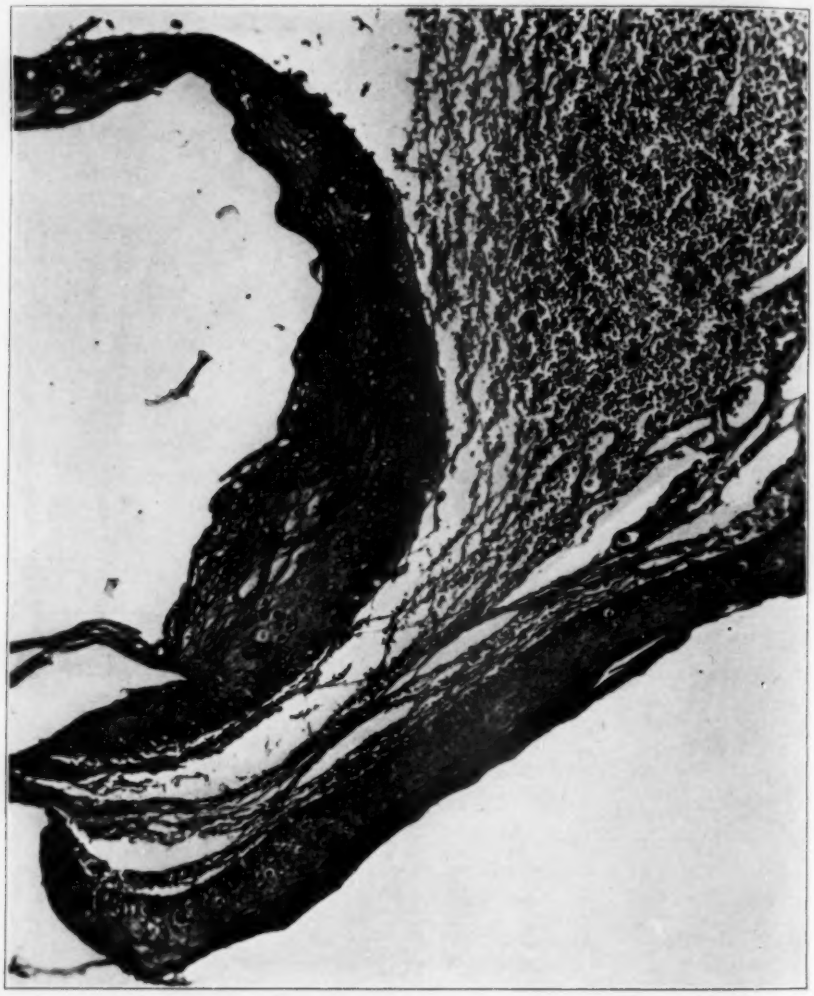

Fig. 2.—Section through the edge of the cyst, to show in detail the character of the wall and both squamous epithelial coverings (× 75).

COMMENT

The most recent work in which cysts of the larynx are discussed is the exhaustive study by New and Erich,[2] who in 1938 reported a series

2. New, G. B., and Erich, J. B.: Benign Tumors of the Larynx: A Study of Seven Hundred and Twenty-Two Cases, Arch. Otolaryng. **28**:841-910 (Dec.) 1938.

of 722 cases of benign laryngeal tumor. Among 25 laryngeal cysts reported by both authors, only 1 was classified as of congenital origin. Later they added 2.[3] No exact figures as to the relative frequency of the congenital type have been available to us. The congenital cyst is usually described as having a stratified columnar epithelial lining. In instances in which the fluid pressure has been great, the epithelial lining may be absent. In our case the cyst was lined by definitely squamous epithelium. This fact, which to us represented a strong diagnostic point differentiating this cyst from the more common retention cyst, may be explained on the basis either of metaplasia or of direct origin of the cysts from laryngeal mucosa. Other characteristic features of the congenital cyst are glandular structures and a specific subepithelial stroma in which lymphocyte aggregations are common. The presence of true lymphoid follicles must be regarded as unusual. Those observed in our case were prominent, with a very cellular and distinct germinal center. These findings may be of interest in that New and Erich [3] used the absence of such follicles in their cases as a basis for refuting the theory of Louys,[4] that the congenital cyst is actually of branchiogenic origin. New and Erich accepted rather the theory of Schneider,[5] that the congenital cyst arises from cells displaced in the formation of the appendix of the ventricle, feeling that definite lymph follicles should be present if the cyst is supposed to be branchiogenic. Although we do not wish, from the evidence presented in 1 case, to discuss the theoretic origins of the laryngeal cyst, we may point out that our observation well conforms with Louys' view.

SUMMARY

A case of congenital laryngeal cyst is reported, in which removal was successful, resulting in complete cure. Pathologically the cyst presents the unusual feature of a stratified squamous epithelial lining with numerous hyperplastic lymph follicles within its wall.

Springfield City Hospital.

3. New, C. B., and Erich, J. B.: Congenital Cysts of Larynx: Report of Case, Arch. Otolaryng. **30**:943-949 (Dec.) 1939.

4. Louys, E., cited by New, G. B.: Cysts of the Larynx, Journal-Lancet **37**:99-102 (Feb. 15) 1917.

5. Schneider, P., cited by New and Erich.[3]

ABERRANT SALIVARY GLAND IN TONSILLAR FOSSA

WILLIAM J. HITSCHLER, M.D., AND THOMAS A. COPE JR., M.D., PHILADELPHIA

An aberrant salivary gland may be of more frequent occurrence than one would believe from the number of cases reported. Only 4 could be found in a survey of the literature, the most recent being that reported by Prohovnik.[1]

REPORT OF A CASE

W. E., aged 4, in the service of Dr. B. D. Parish, was referred for tonsillectomy on account of earaches and cough. Her tonsils were of moderate size. They were removed by dissection with the patient under ether anesthesia. After the right tonsil was removed by means of a snare, there appeared at the lower part of the fossa a pinkish white tissue, which at first was thought to be unremoved tonsillar tissue. Closer examination showed it to be smooth and soft with a firm surface. It resembled a thick-walled cyst or a large blood vessel. It seemed to be attached laterally to the muscular bed of the fossa either by a small area or by a short pedicle. There was no pulsation in it; hence it was deemed safe to remove. The feel of the snare in removing the growth was of cutting through fibrous tissue. The tissue came out completely, without bleeding and without leaving any trace. Even its attachment could not be seen. The growth did not extend through the muscle.

The pathologic report stated that the gross specimen consisted of two partially encapsulated pieces of pale mucoid tissue 18 by 13 by 8 mm. and 15 by 10 by 3 mm. Microscopic sections showed a small, well formed salivary gland covered by a fibrous capsule and consisting chiefly of mucus-secreting acini. The pathologic diagnosis was aberrant salivary gland.

Two of the observers of the 4 cases previously reported stated that the tissue found in the tonsillar fossa was lobulated. Two said that this tissue was soft, and two that it was yellowish gray. One said the growth grossly resembled a typical salivary gland. In 3 of the 4 cases the growth was found in the lower part of the fossa, and in the fourth it was in the upper part. One was the size of a "prune stone;" another measured probably 1.5 cm. in each direction. Two authors stated that the tissue extended out into the neck through the constrictor muscle. In 3 cases the tissue was not removed, save a specimen for biopsy, and in the fourth it was removed. The reason for not removing it in 1 case was the proximity of the great vessels and nerves. No reason was given in the other cases.

Microscopically 1 growth was described as parotid gland tissue, 1 as a typical salivary gland, 1 as a submaxillary gland and 1 as a mixed salivary

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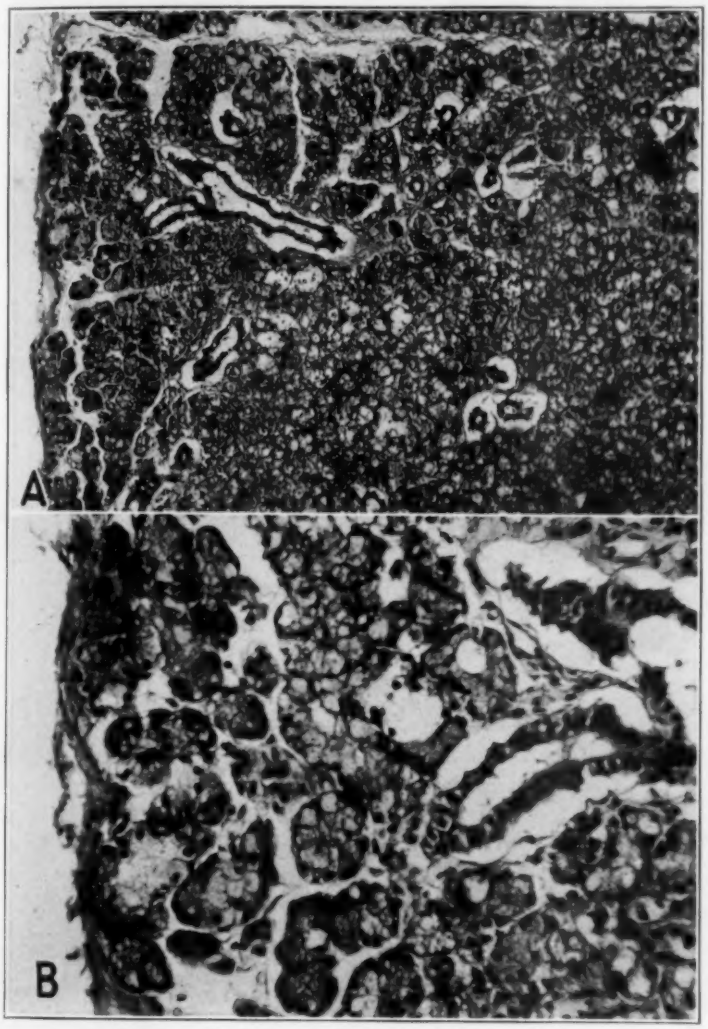
Presented before the Section on Otolaryngology of the College of Physicians, Philadelphia, Oct. 16, 1940.

1. Prohovnik, J.: Salivary Gland Tissue in the Tonsillar Fossa, Ann. Otol., Rhin. & Laryng. **45**:1181-1183 (Dec.) 1936.

gland with mucous alveoli predominating. The salivary glands are composed of serous and mucus-secreting elements in varying proportion. In the parotid gland the former are preponderant. In the other glands the mucus-secreting elements form a greater proportion. Of the reported



(*A*) Low power and (*B*) high power photomicrographs, showing glandular structure and capsule.

cases, we group together the first 2, in which the tissue consisted largely of serous acini, and add our own to the last 2, in which the growth was composed chiefly of mucous elements. A capsule was not mentioned in

any previously reported case. The absence of a capsule probably accounts for the difference in appearance and consistency of the structure here reported.

NOTE.—In the discussion following the presentation, Dr. Warren B. Davis said that he had encountered 3 cases in which there was salivary gland tissue in the tonsillar fossa, and Dr. Joseph C. Donnelly showed a slide of tissue taken from a patient of his.

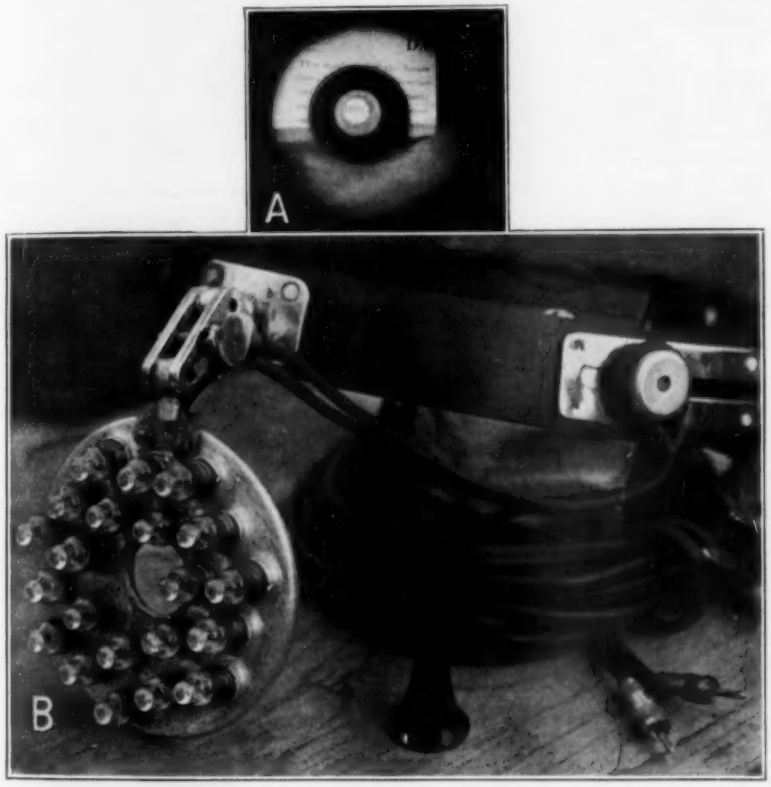
Clinical Notes

A NEW HEADLIGHT

LEWIS B. SASLAW, M.D., STOCKTON, CALIF.

For speculum, or "keyhole," work the head mirror and a separate source of light comprise the basic means of illumination.

However, from the standpoint of convenience the head mirror has certain disadvantages—the principal one is that the relationship between the source of



A, print illuminated by the headlight. The camera was focused through an ear speculum. *B*, the headlight.

light, the observer's head, the position of the mirror and the object to be illuminated must be kept constant. A change in position of one of these necessitates an adjustment of one or more of the other factors.

The light here presented represents an attempt to approximate the good qualities of the head mirror and at the same time eliminate some of the disadvantages.

The concave head mirror in operation becomes a multiple point source of light, which is directed at an object. The illuminated object is viewed through the beam of light.

The light here described has been constructed on this basic principle. It consists of multiple series of bulbs arranged around a central viewing aperture. In construction it is simple. Two metallic plates are separated by an insulating disk. The front plate is threaded to receive ordinary prefocused flashlight bulbs. The back plate makes contact with the tips of the bulbs. The hole in the center is approximately ¾ inch (1.9 cm.) in diameter, giving a large field of vision with a minimum of manipulation of the light. The bulbs are obtainable in most drugstores and are supplied by a transformer. They operate independently, and failure of one or several is of no consequence.

The photographs are self explanatory. *A* was made with the camera behind the light, focused through an ear speculum on the print, which is illuminated by the headlight.

SUMMARY

A new light is described, which simulates a head mirror in operation and eliminates the disadvantages of a mirror.

It is easily manipulated, and the large aperture gives a large field of vision with a minimum of manipulation.

The construction is simple, and the bulbs are inexpensive and easily obtained.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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PLASTIC SURGERY, 1940

ROBERT H. IVY, M.D., D.D.S.
AND
HENRY A. MILLER, M.D., D.D.S.
PHILADELPHIA

Owing to the war, many foreign publications were not available for abstracting. A marked increase in articles on military surgery was noted; the literature in this field pertaining to plastic surgery is included in this review.

Outstanding achievements during the year were the further development of sulfanilamide and its derivatives for use in the prevention and treatment of wound infections, the introduction of a noncapillary, nonirritating suture material made of synthetic plastics, the demonstration of the feasibility of using large calibrated skin grafts cut with the dermatome and the acceptance on a practical clinical basis of the use of preserved cartilage for implants to restore facial and nasal contours.

The literature on plastic surgery for 1940 can be conveniently grouped under five headings: (1) results of clinical and laboratory research—general principles; (2) plastic operations on the face; (3) plastic operations on the nose; (4) plastic operations on the ears; (5) plastic operations on the jaws and palate—surgical intervention in war wounds.

RESULTS OF CLINICAL AND LABORATORY RESEARCH—GENERAL PRINCIPLES

The introduction of synthetic plastics as suture material stimulated renewed interest in the entire problem of sutures and tissue reactivity. Comparison of catgut, silk, linen and cotton by Meade and Ochsner [1] showed that cotton and silk have similar good qualities. In an article on the silk technic, Shambaugh [2] demonstrated that lubrication with petrolatum was desirable to prevent fraying and breaking of silk when

1. Meade, W. H., and Ochsner, A.: The Relative Value of Catgut, Silk, Linen and Cotton as Suture Materials, Surgery **7**:485-513 (April) 1940.
2. Shambaugh, P.: The Silk Technique, Surgery **7**:9-23 (Jan.) 1940.

knots are tied under tension. Studies of nylon and silk by Nichols and Diack [3] showed that there is little reaction to either suture material, that both are superior in this respect to catgut and that in routine operating nylon is easier to handle and more uniform in strength than silk. Bellas [4] reported superior results with synthetic plastic suture.

Despite meticulous technic, clean surgical wounds will occasionally become infected. In a clinical study, Casberg [5] definitely noted that implantation of sulfanilamide in a wound was well tolerated and beneficial. Sheehan [6] described his excellent results with powdered sulfanilamide in the treatment and prevention of sepsis in war wounds.

In investigations of maggot therapy of infected wounds, ammonium bicarbonate was noted to be present. Robinson [7] reported that 1 to 2 per cent sterile solution of ammonium bicarbonate as a wet dressing was efficacious in combating infection. Johnson's [8] studies of antiseptics for use in contaminated wounds suggested the superiority of azochloramid.

The general scope of plastic surgery was discussed by Updegraff,[9] Gingrass [10] and Schaefer.[11] A review of the literature was contributed by McDowell and Brown.[12]

The indications for skin grafting, according to Elis and Von Wedel,[13] include skin losses due to burns or accidental trauma, postoperative skin defects, acute necrotizing infections, congenital or acquired blemishes, ulcers due to various causes, loss of mucous membrane and lack of part or all of an organ, such as the nose or the auricle. The use of pinch grafts for covering raw surfaces is justified only by the high per cent

3. Nichols, H. M., and Diack, A. W.: An Experimental Study of Nylon as a Suture Material, West. J. Surg. **48**:42-46 (Jan.) 1940.

4. Bellas, J. E.: Suture Studies: A New Suture, Arch. Surg. **41**:1414-1426 (Dec.) 1940.

5. Casberg, M. A.: Sulfanilamide Implantation as a Method of Controlling Infection in Clean Surgical Wounds, J. Missouri M. A. **37**:473-474 (Nov.) 1940.

6. Sheehan, J. E.: Wounds in Modern War, M. Rec. **152**:165-167 (Sept. 4) 1940.

7. Robinson, W.: Ammonium Carbonate Secreted by Surgical Maggots Stimulates Healing in Purulent Wounds, Am. J. Surg. **47**:111-115 (Jan.) 1940.

8. Johnson, P.: Wound Healing, West. J. Surg. **48**:415-423 (July) 1940.

9. Updegraff, H. L.: Reparative Surgery, Australian & New Zealand J. Surg. **9**:237-258 (Jan.) 1940.

10. Gingrass, P.: Plastic Surgery of the Face, Am. J. Orthodontics **26**:961-968 (Oct.) 1940.

11. Schaefer, J. E.: Oral and Plastic Surgery, J. Indiana M. A. **30**:60-66 (Feb.) 1940.

12. McDowell, F., and Brown, J. B.: A Review of Reconstructive Surgery of the Face, Laryngoscope **50**:1117-1138 (Dec.) 1940.

13. Ellis, S. S., and Von Wedel, C.: The Indications, Results and Reasons for Use of the Different Types of Skin Grafts, J. Oklahoma M. A. **33**:8-15 (Dec.) 1940.

of successful applications. Davis [14] recommended the small deep graft for uniformly satisfactory results in the repair of surface defects. Split grafts are successful in over 90 per cent of cases and can be used for extensive areas. About 15 per cent contraction occurs. Full thickness grafts are to be used for weight-bearing surfaces; there is about 40 per cent contraction. Where more tissue is needed, a pedicle graft is of value. In analyzing failures with Thiersch grafts, Rank [15] mentioned sepsis, hematoma and inadequate pressure fixation as the chief causes. Management of the preoperative and postoperative phases of skin grafting was outlined by Havens.[16] Keyes [17] reported the replacement of a completely avulsed piece of skin from the dorsum of a finger with complete success. In an interesting case report, Harrell and Valk [18] described autogenous transplantation of a fibrosarcoma during application of a full thickness graft. For migration of a pedicle flap, temporary attachment of one end of the tube to a neighboring area may be necessary in order to allow the other end to be rotated up into the wound to be grafted. To permit good approximation of the tube to the recipient area, with only negligible scarring after the tube is removed, Claoué [19] used a circular incision or a crossed incision on the recipient area for best results. Successful transplantation of complex structures, as the toe to the finger, were reported by Neuhof [20] and by Blair and Byars.[21]

Wound closure with avoidance of scars can be achieved satisfactorily by certain technics. González Ulloa [22] favors subcuticular suturing. Updegraff [23] uses the same method but stresses early removal of sutures, wound support with adhesive straps and pressure dressings to prevent hematoma.

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14. Davis, J. S.: The Use of Small Deep Grafts in the Repair of Surface Defects, Am. J. Surg. **47**:280-298 (Feb.) 1940.

15. Rank, B. K.: Use of the Thiersch Skin Graft, Brit. M. J. **1**:847-849 (May 25) 1940.

16. Havens, F. Z.: Preoperative and Postoperative Care in Skin Grafting, S. Clin. North America **20**:1087-1092 (Aug.) 1940.

17. Keyes, E. L.: Graft in Situ of Skin Completely Avulsed, J. Missouri M. A. **37**:75-76 (Feb.) 1940.

18. Harrell, G. T., and Valk, A. de T.: Autogenous Transplantation of a Fibrosarcoma During Application of a Full-Thickness Skin Graft, Ann. Surg. **111**:285-291 (Feb.) 1940.

19. Claoué, C.: Lambeaux cutanés tubulés transportés par inclusion dermique ou procédé de la sangsue, Monde méd. **50**:149-152 (May) 1940.

20. Neuhof, H.: Transplantation of Toe for Missing Finger: End Result, Ann. Surg. **112**:291-296 (Aug.) 1940.

21. Blair, V. P., and Byars, L. T.: Toe to Finger Transplant, Ann. Surg. **112**:287-291 (Aug.) 1940.

22. González Ulloa, M.: Tratamiento quirúrgico de las cicatrices, Rev. mex. de cir. ginec. y cáncer **8**:321-329 (Aug.) 1940.

23. Updegraff, H. L.: Wound Closure, with Particular Reference to the Avoidance of Scars, Am. J. Surg. **50**:749-754 (Dec.) 1940.

When scars are present, they can often be improved by surgical intervention. The tissues, according to May,[24] must have reached a state of quiescence before operation is undertaken. May described the methods he used in the management of scars, particularly those which are contracted and depressed. Examination to determine why scars are painful revealed the presence of neuromas at the points of tenderness. Kredel [25] found that relief of symptoms followed excision of the painful areas alone; it was not necessary to excise the entire scarred area. Extensive scarred areas may require more than simple excision and yet not call for skin grafting. Zeno [26] described the various methods available, such as the use of the Z plastic, tissue rotation and sliding flaps, which utilize neighboring skin to cover the defect made by excising the scars. Plastic surgery often requires meticulous and delicate handling of the parts, and in order to secure better visualization of fine details, MacCollum [27] devised a loupe which enables comfortable magnification of the entire operative field.

Burns of the skin may be due to thermal, chemical, electric and roentgenologic agents. Nagle [28] recommended an antiseptic local anesthetic for the management of extensive burns. Edwards and Bowie [29] stated that the destruciton of tissue in electric burns is due to the heat generated in the tissues. In their management, the conservative method of permitting nature to build up new tissue beneath and around the destroyed tissues usually leads to the most satisfactory results.

An unfortunate sequela of roentgen ray and radium burns is the development of indurated, boardlike skin which ulcerates, is painful and may lead to deformity and loss of function. Ghormley and Fairchild [30] found that about 20 per cent of such burns also undergo malignant change. Excision of the scarred and ulcerated area followed by skin grafting yields good results. The most generally suitable type of graft is the split thickness graft, although there may be indications for any of the commonly used grafts in special cases.

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24. May, H.: Correction of Scars, Am. J. Surg. **50**:754-761 (Dec.) 1940.

25. Kredel, F. E.: Painful Scars, Surgery **8**:98-105 (July) 1940.

26. Zeno, L.: Mano en garra. Plástica de Morestin combinada con injerto libre de piel total, An. de cir. **6**:315-321 (Sept.) 1940; Cicatrices retráctiles de la región cérvico-facial, ibid. **6**:341-349 (Dec.) 1940.

27. MacCollum, D. W.: A Magnified Operative Field, Am. J. Surg. **49**:528-530 (Sept.) 1940.

28. Nagle, P.: Use of Antiseptic Anesthetic Agent Locally in Extensive Burn, J. Oklahoma M. A. **33**:14-16 (Jan.) 1940.

29. Edwards, C. R., and Bowie, H. C.: High Tension Electric Burns, Am. J. Surg. **47**:299-311 (Feb.) 1940.

30. Ghormley, R. K., and Fairchild, R. D.: Surgical Treatment of Roentgen and Radium Dermatitis, Surgery **7**:736-752 (May) 1940.

PLASTIC OPERATIONS ON THE FACE

Foster [31] presented the principles of diagnosis and the indications for treatment which should be routinely followed in cases of injury of the face. Any injury of the face warrants an examination of the eyes sufficient to permit observation of function, visualization of the fundus and gross estimation of intraocular pressure. In examination of the nose, air in the subcutaneous tissues may suggest to the examiner that the paranasal sinuses have been injured. Such a condition should be treated as potentially serious because of the possibility of meningitis. In examination of the face, all the facial functions must be considered and any alterations corrected as far as possible. Future correction is greatly obviated by careful and competent first treatment.

According to New and Erich,[32] proper care of the primary wound of the face includes debridement, complete hemostasis, adequate drainage and prevention of hematoma. In severe lacerations of the face, the seventh nerve and the parotid duct may be cut. If the main branch of the nerve is cut, the ends should be sutured. If a peripheral branch is cut, some regeneration will probably occur. Opening of the parotid duct may result in salivary fistula. They have successfully used the method of Glascock, in which strands of silkworm gut are threaded through the cut ends of the duct as scaffolding for repair.

Erich [33] feels that a significant advance in the management of large losses of skin has been made by the immediate application of free skin grafts to these raw surfaces that are not grossly contaminated. The best matching skin is that taken from the postauricular area.

Swelling of the soft tissues due to trauma may mask an underlying fracture of the zygoma. Any irregularity of the orbital rim or any unexplained difficulty in opening the mouth which may be due to impingement of the fractured bone on the coronoid process of the mandible should be investigated roentgenologically. Kanthak [34] reviewed the various methods advanced for reduction of these fractures. In the 26 successful cases which he reported, he used either the Gillies temporal approach or elevation of the fragment through a slit in the buccal mucosa on the affected side.

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31. Foster, A. K., Jr.: Severe Injuries of the Face, Am. J. Surg. **48**:391-397 (May) 1940.

32. New, G. B., and Erich, J. B.: The Care of Automobile Injuries Involving the Face, Minnesota Med. **23**:1-8 (Jan.) 1940.

33. Erich, J. B.: Traumatic Facial Injuries, Surg., Gynec. & Obst. **71**:822-824 (Dec.) 1940.

34. Kanthak, F. F.: Fractures of the Zygomatic Bone, Surgery **7**:796-805 (May) 1940.

Unexplained flattening of the face may occur about the fifteenth year and progress to a generalized facial hemiatrophy. Kazanjian and Sturgis [35] reported 6 cases in which they used dermal, fat and fascia grafts to fill out the contours of the affected side of the face.

Mason, Anson and Beaton [36] reported a case of double lower lip and advanced an embryologic explanation of the occurrence of this abnormality. Surgical correction consisted of an elliptic excision to the depth of the sulcus to produce a normal lip.

For loss of a lower lip following an infection, Cunningham [37] devised a simplified operation. The labiogingival sulcus was first lined with a free skin graft, which was stabilized in place with a modeling compound splint. To close the defect, a lining was turned up from the neighboring skin, while the outer cover was supplied by a pedicle graft from the cheek.

Forehead defects may be congenital (due to persistence of the frontal suture), postoperative (secondary to intracranial or extracranial operative procedures), osteomyelitic or traumatic. Writing on this subject, Peer [38] and New and Dix [39] recommended dermal grafts for small defects and bone and cartilage implants as useful in larger defects.

For osteomyelitis of the frontal bone, Kazanjian and Converse [40] recommended the inverted T incision with rounded corners as the most satisfactory approach from the point of view of plastic surgery. Following operation, a delay of three months was found necessary before plastic closure could be undertaken. The first step consisted in bringing the skin flaps down from their position curled up over the edge of the bone. Postoperative roentgenographic studies of 18 patients revealed regeneration of bone in only 2, both children. The second stage consisted in using osteoperiosteal grafts from the tibia to fill out the defect.

The indications and principles of the surgical correction of blepharoptosis were presented by Kirby.[41] With regard to paralysis of the

35. Kazanjian, V. H., and Sturgis, S. H.: Surgical Treatment of Hemiatrophy of the Face, J. A. M. A. **115**:348-354 (Aug. 3) 1940.

36. Mason, M. L.; Anson, B. J., and Beaton, L. E.: The Surgical and Anatomical Aspects of a Case of Double Lower Lip, Surg., Gynec. & Obst. **70**:12-15 (Jan.) 1940.

37. Cunningham, A. F.: Reconstruction of Lower Lip, Northwest Med. **39**: 336-337 (Sept.) 1940.

38. Peer, L. A.: Types of Buried Grafts Used to Repair Deep Depressions of the Skull, J. A. M. A. **115**:357-360 (Aug. 3) 1940.

39. New, G. B., and Dix, C. R.: Repair of Defects of the Frontal Bone, Surg., Gynec. & Obst. **70**:698-701 (March) 1940.

40. Kazanjian, V. H., and Converse, J. M.: Reconstruction After Radical Operation for Osteomyelitis of the Frontal Bone, Arch. Otolaryng. **31**:94-113 (Jan.) 1940.

41. Kirby, D. B.: Blepharoptosis: The Technique of Its Surgical Correction, Surg., Gynec. & Obst. **70**:438-447 (Feb.) 1940.

lower lid, Blair and Byars [42] described various methods of bringing the lid up closer to the eyeball. These authors also reported their success with the correction of scleral scars by the use of skin grafts applied to the denuded area on a cotton or wax mold. For plastic restoration of a completely exenterated orbit, Hagedoorn [43] employed a four stage operation consisting first in bringing fat in a basal layer all around the orbit, and then following with skin grafting so as to receive an artificial eye.

Correction of facial paralysis due to injury of the main branch of the seventh nerve is achieved quite satisfactorily by nerve suture. Martin [44] reported 16 cases, in 15 of which the results were satisfactory. In cases of intracranial severance of the nerve, anastomosis of the nerve with the hypoglossal is feasible if judged by Coleman's [45] results. Atrophy of the tongue on one side does not appear to carry any particular discomfort to the patient. The classic method of using fascial strips to hold the paralyzed side of the face in symmetry during repose may be aided by a new instrument devised by Macey [46] for passing portions of tendon and fascia.

PLASTIC OPERATIONS ON THE NOSE

Indications for nasal plastic operations were discussed by England.[47] Rhinoplasty may be indicated in the very young, according to Ide.[48] The use of preserved cartilage now allows operations on young persons which were not possible formerly.

In preoperative studies of facial and nasal deformities and for records, life masks are valuable. Peluse [49] described the method of making such masks of latex rubber.

Presumptive signs of fracture of the nasal bones, according to Moore and Harris,[50] are pain, epistaxis, nasal obstruction, ecchymosis, bony

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42. Blair, V. P., and Byars, L. T.: Paralysis of the Lower Lid and Scleral Scars and Grafts, Surg., Gynec. & Obst. **70**:426-437 (Feb.) 1940.

43. Hagedoorn, A.: Plastic Restoration of a Deformity Caused by Complete Exenteration of the Orbit, Surg., Gynec. & Obst. **70**:193-195 (Feb.) 1940.

44. Martin, R. C.: Recent Experiences with Operation on the Facial Nerve. Arch. Otolaryng. **31**:1071-1075 (Dec.) 1940.

45. Coleman, C. C.: Results of Facio-Hypoglossal Anastomosis in the Treatment of Facial Palsy, Ann. Surg. **111**:958-967 (June) 1940.

46. Macey, H. B.: A New Instrument for Passing Portions of Tendons and Fasciae Latae, Am. J. Surg. **47**:686 (March) 1940.

47. England, M. C.: Nasal Plastic Operations, J. Oklahoma M. A. **33**:10-12 (Jan.) 1940.

48. Ide, D.: Rhinoplasty: Its Social Side, Eye, Ear, Nose & Throat Monthly **29**:174-176 (July) 1940.

49. Peluse, S.: A Simplified Technic for the Construction of Life Masks of Latex Rubber, Arch. Otolaryng. **31**:955-966 (July) 1940.

50. Moore, P. M., and Harris, H. E.: Fracture of the Nasal Bones, Am. J. Surg. **50**:668-672 (Dec.) 1940.

crepitus and possibly emphysema. Roentgenograms should be taken for assistance in diagnosis and for records. As to the immediate care of the traumatized nose, Kimball and Drummond [51] recommended that no fragments be removed unless they are completely detached. In simple fracture, if splinting is necessary, fine steel wire mattressed through the bridge and fixed over lead plates may be used. For a more severe injury, internal splints in the nose and attached to the teeth or a head cap for stability may be necessary.

Small losses of skin about the nasal tip, according to Straatsma,[52] are usually repaired by free grafts from the eyelids or postauricular area because of the excellent matching of the skin. As to larger losses, the ala nasi is repaired by turning down a hinge flap from the tissue surrounding the deformity and overlaying that with a skin graft for outer coverage. Twyman [53] simplified this procedure by splitting the neighboring ala nasi and sliding the outer layer over for coverage. Ivanissevich and Ferrari [54] described the transfer of a full thickness of a portion of the auricle to restore nasal alar defects.

In analyzing hump nose deformities, Brown [55] recommended careful preoperative study. In many cases, additional procedures, such as shortening the nose and narrowing the dorsum, may be necessary in order to secure the best esthetic result.

In correcting deviations of the nose, the classic procedure is to remove a triangular piece of bone on the wide or convex side to allow reposition of the nose in the midline. Sarnoff [56] eliminated this step by removing the dorsal hump at different levels to compensate for the wide side.

The correction of a nasal deformity may improve both appearance and function. A number of structural abnormalities of the nares may be surgically corrected with expected improvement in aeration. Cinelli [57] discussed these deformities and added the condition of alar collapse due to muscular imbalance or atrophy of the muscles around the nares. His correction of this condition consisted of lateral placement of the ala in a fixed open position.

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51. Kimball, G. H., and Drummond, N. R.: Traumatic Affections of the Nose, J. Oklahoma M. A. **33**:1-3 (April) 1940.

52. Straatsma, C. R.: Some Problems in Nasal Plastic Surgery, Laryngoscope **50**:1092-1099 (Nov.) 1940.

53. Twyman, E. D.: Partial Defects of the Nose, West. J. Surg. **48**:106-109 (Feb.) 1940.

54. Ivanissevich, O., and Ferrari, R. C.: Rhinoplasty: Argentine Method, Surg., Gynec. & Obst. **71**:181-187 (Aug.) 1940.

55. Brown, A. M.: Plastic Operations for Hump Nose, Arch. Otolaryng. **31**: 827-838 (May) 1940.

56. Sarnoff, J. A.: Simplified Plastic Operation for Hump, Hook and Twist of the Nose, Surgery **7**:908-909 (June) 1940.

57. Cinelli, A. A.: Alterations in Nasal Function Due to Anatomic Variations of the Nares, Arch. Otolaryng. **31**:53-64 (Jan.) 1940.

Deformities due to septal abnormalities are chiefly caused by deviation or dislocation of the cartilaginous septum from the maxillary crest during childhood with subsequent distortion of the alar cartilages with age. Kopp [58] corrected these deformities by relieving both the septum and the alar cartilages and repositioning them in the midline. Harbert [59] described a simplified operation to replace the laterally displaced free border of the septal cartilage. In discussing whether atrophic rhinitis is a contraindication for contemplated plastic operation on the nose, Kopp [60] noted that patients with such a condition often showed improvement following operations, particularly after operations which narrowed the nose.

Rhinoplastic operations require surgical skill and artistic ability. Safian [61] and Maliniac [62] presented papers illustrating the pitfalls and errors one may fall into if one undertakes such an operation without being fully trained for it.

Saddle nose deformities are corrected by a variety of procedures. According to Goldman,[63] minor defects may be easily corrected by the use of septal cartilage removed at submucous resection. Harbert [64] recommended a specially cut piece of cartilage taken from the auricle. Sarnoff [65] described methods of raising the bridge of the nose without the use of implants. Brown [66] favored using the patient's own cartilage to fill out the depression. Cohen [67] favored an osteocartilaginous graft which allows good contouring and will not absorb; union with the nasal bones will be firm in time. This graft offers a special advantage in

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58. Kopp, M. M.: Nasal Deformities Due to Septal Abnormalities, M. Rec. **151**:111-116 (Feb. 21) 1940.

59. Harbert, F.: Correction of Lateral Displacement of Free Border of the Septal Cartilage, Arch. Otolaryng. **31**:341-343 (Feb.) 1940.

60. Kopp, M. M.: Atrophic Rhinitis in Plastic Surgery, Laryngoscope **50**:510-519 (June) 1940.

61. Safian, J.: Failures in Rhinoplastic Surgery, Am. J. Surg. **50**:274-281 (Nov.) 1940.

62. Maliniac, J. W.: Prevention and Treatment of Late Sequelae in Corrective Rhinoplasty, Am. J. Surg. **50**:84-91 (Oct.) 1940.

63. Goldman, I. B.: Correction of Dorsal Depressions by Septal Implants, Arch. Otolaryng. **32**:524-529 (Sept.) 1940.

64. Harbert, F.: Correction of Saddleback Deformities of the Nose by Specially Cut Cartilage from the Ear, Arch. Otolaryng. **31**:339-341 (Feb.) 1940.

65. Sarnoff, J.: Procedure of Choice in Correction of Saddle Nose, M. Rec. **151**:155-156 (March 6) 1940.

66. Brown, J. B.: Preserved and Fresh Homotransplants of Cartilage, Surg., Gynec. & Obst. **70**:1079-1082 (June) 1940.

67. Cohen, L.: Advantages of Mixed Bone and Cartilage Grafts in Correction of Saddle Nose and Other Depressed Defects of the Dorsum, Ann. Otol., Rhin. & Laryng. **49**:410-417 (June) 1940.

those cases in which the tip is moderatley fallen, as an intraseptal support is often unnecessary when this method is used and firm union of the part is obtained.

Saddle nose deformity due to syphilis requires a much more complex restoration. Cinelli [68] described a one stage procedure of freeing the nose up through the gingivolabial sulcus and then, with new gowns and gloves and a new sterile instrument tray, proceeding with the second graft of the operation, in which cartilage is inserted to build out the nose.

New instruments for rhinoplasty were described by Pearlman [69] and by Cinelli.[70] Aufricht [71] presented a modeling compound splint which he has found very serviceable. Brown [72] devised an excellent tissue forceps.

Congenital atresia of the posterior part of the nares is usually treated by removal of the obstruction and of a portion of the posterior part of the vomer. Mangiaracina [73] reported his successful use of this operation. Where the tissue was not too severely scarred, Goodyear [74] made a horizontal incision into the nasopharynx and kept it open with a loop of rubber catheter introduced through the nostrils. Morgenstern [75] used electrocoagulation on this type of patient with excellent results.

PLASTIC OPERATIONS ON THE EAR

A classification of ear deformities was presented by Foucar.[76] Often a deformed ear is associated with a stenosed external auditory canal, and the question is raised whether a bony canal is present. Camp and Allen [77] found tomography valuable in visualization of the canal. For

68. Cinelli, A. A.: The Syphilitic Nose, Laryngoscope **50**:520-526 (June) 1940.

69. Pearlman, L. M.: Improved Instruments and Postoperative Splint for Nasal Plastic Operations, Arch. Otolaryng. **32**:338-340 (Aug.) 1940.

70. Cinelli, J. A.: New Instruments for Use in Rhinoplastic Surgery, Arch. Otolaryng. **31**:1102-1107 (Dec.) 1940.

71. Aufricht, G.: Dental Molding Compound Cast and Adhesive Strapping in Rhinoplastic Surgical Procedure, Arch. Otolaryng. **32**:333-338 (Aug.) 1940.

72. Brown, J. B.: New Type of Tissue Forceps, Am. J. Surg. **49**:397 (Aug.) 1940.

73. Mangiaracina, A.: Congenital Atresia of Posterior Nares, Arch. Otolaryng. **32**:1088-1089 (Dec.) 1940.

74. Goodyear, H. M.: Nasopharyngeal Atresia: Description of an Operation, Arch. Otolaryng. **32**:522-523 (Sept.) 1940.

75. Morgenstern, D. J.: Congenital Atresia of the Postnasal Orifices, Arch. Otolaryng. **31**:653-662 (April) 1940.

76. Foucar, H. O.: Congenital Abnormalities of the External Ear, Canad. M. A. J. **43**:26-27 (July) 1940.

77. Camp, J. D., and Allen, E. P.: Microtia and Congenital Atresia of the External Auditory Canal, Am. J. Roentgenol. **43**:201-203 (Feb.) 1940.

reconstruction of the ear, Kirkham [78] used preserved ear cartilage successfully. Protruding ears are the result of a congenital malformation of the anthelix. New and Erich [79] recommended that the protrusion be corrected at about 5 years of age, before the child goes to school so as to preclude development of any emotional disturbance as a result of his abnormality.

Postauricular defects usually follow secondary infection of a wound in the mastoid process of the temporal bone. To correct such a defect, Greenfield [80] first eradicates all vestige of diseased bone and granulations in the mastoid process. The size of the bony defect is diminished by lowering the posterior canal wall, removing widely the tip of the mastoid process and leveling off all surrounding bone. For closure the incision over the mastoid is extended to expose the temporal muscle, from which a flap is cut and turned down into the cavity. The scar is excised, and the wound closed in layers by splitting the flap into an outer and an inner flap. A posterior relaxation incision is made if necessary. For large defects O'Brien and Slaughter [81] described an operation based on anatomic studies. Their muscle flap operation utilizes the posterior branch of the middle temporal artery for nourishment, so that a sufficiently large flap can be turned down to fill out a large defect and restore contour. After closure they use a special modeling compound pressure dressing. Fifteen cases are reported; all the patients had previously undergone operation, and in all the muscle flap operation was used successfully.

PLASTIC OPERATIONS ON THE JAWS AND PALATE—SURGICAL INTERVENTION IN WAR WOUNDS

Cole,[82] Beltrami [83] and Bercher [84] all stressed the need of specialist management of maxillofacial injuries for best results. First aid care, however, will do much to advance subsequent repair. According to

78. Kirkham, H. L. D.: The Use of Preserved Cartilage in Ear Reconstruction, Ann. Surg. **111**:896-901 (May) 1940.

79. New, G. B., and Erich, J. B.: Protruding Ears: A Method of Plastic Correction, Am. J. Surg. **48**:385-390 (May) 1940.

80. Greenfield, S. D.: Operation for the Cure of Postauricular Fistulae, Laryngoscope **50**:312-325 (April) 1940.

81. O'Brien, G. R., and Slaughter, W. B.: A Method for Reconstruction of Postauricular Defects, Arch. Otolaryng. **31**:65-73 (Jan.) 1940.

82. Cole, P. P.: War Injuries of Jaws and Face, Post Graduate M. J. **16**:233-246 (July) 1940.

83. Beltrami, G.: Conduite à tenir dans les fractures des maxillaires, Marseille-méd. **1**:32-37 (Jan. 15) 1940.

84. Bercher, J.: Comment assurer, pour les polyblessés, une liaison efficace entre le chirurgien maxillo-facial et les autres chirurgiens, Rev. de stomatol. **41**: 759-760, 1940.

Garnett,[85] Lockwood,[86] Stout [87] and Davis,[88] there should be immediate debridement and primary suture with drainage and the prevention of hemorrhage, sepsis and deformity. For gaping injuries around the mouth with loss of substance, skin should be sewn to mucous membrane to close all raw areas possible.

Injuries to the teeth and jaws can be handled by a primary apparatus as described by Fairbank and Ivy.[89] The aim of all such treatment is to maintain the occlusion of the teeth and buccal sulcus intact. Current methods of management of fractured jaws utilize intraoral fixation when possible, and extraoral traction when needed. These methods were described by Frizell,[90] Ponroy and Psaume,[91] McDonough,[92] Davidson and Brown [93] and Woodward.[94] Management of the fractured parts by the use of vitallium screws placed through the skin was reported by Bigelow.[95] Cameron [96] found that vitallium was the least irritating of the metals used in bone management. For cases of nonunion of the mandible, Cole [97] used a pedicle bone graft from the mandible for bridging over the defect with success.

For prognathism of the lower jaw, Haynen [98] has reviewed the Gigli saw method of setting of the jaw back in better occlusion. A congenital

85. Garnett, E. R.: Notes on the Immediate Treatment of Wounds of the Face Preparatory to Plastic Repair, J. Roy. Nav. M. Serv. **26**:273-275 (July) 1940.

86. Lockwood, A. L.: Surgical Problems in War, Brit. M. J. **1**:494-495 (March 23) 1940.

87. Stout, R. A.: Gunshot Wounds of the Face and Jaws: First Aid Treatment, Field Service, Mil. Surgeon **87**:247-250 (Sept.) 1940.

88. Davis, E. D. D.: Treatment and Primary Suture of Face Wounds, Brit. M. J. **1**:381-382 (March 9) 1940.

89. Fairbank, L. C., and Ivy, R. H.: Emergency Treatment and Primary Apparatus for Fractures of Jaws in Warfare, Mil. Surgeon **86**:124-134 (Feb.) 1940.

90. Frizell, C. L.: Fractures of the Mandible, South African Dent. J. **14**: 115-123 (May) 1940.

91. Ponroy and Psaume: Du choix des appareils de réduction et de contention dans les fractures des mâchoires, Rev. de stomatol. **41**:782-789, 1940.

92. McDonough, F. J.: Treatment and Complications in Fractures of the Maxillary Bones, Am. J. Orthodontics **26**:162-175 (Feb.) 1940.

93. Davidson, J. B., and Brown, A. M.: The Management of Fractures of the Maxilla, Mil. Surgeon **87**:26-42 (July) 1940.

94. Woodward, C. M.: Devices for Reduction of Maxillary Fractures, Mil. Surgeon **87**:525-531 (Dec.) 1940.

95. Bigelow, H. M.: The Treatment of Fractures of the Mandible with Vitallium Screws, M. Bull. Vet. Admin. **17**:54-56 (July) 1940.

96. Cameron, D. M.: Vitallium in Bone Surgery with Particular Reference to Its Clinical Irritability, Proc. Staff Meet., Mayo Clin. **15**:561-564 (Sept. 4) 1940.

97. Cole, P. P.: Pathological Fracture of the Mandible: Nonunion Treated with Pedicled Bone Graft, Lancet **1**:1044-1045 (June 8) 1940.

98. Haynen, A. S.: Mandibular Resection, Am. J. Orthodontics **26**:793-796 (Aug.) 1940.

absence of the mandible was corrected by Kazanjian [99] by a transplant of bone extending from the temporal bone above to the mandible below. When union was firm, an arthoplasty was performed on the upper end. Temporomandibular ankylosis treated by the external approach was the subject of a paper by Bellinger.[100] New and Erich [101] in an excellent monograph discussed the problem of retruded chins and methods of correction. Ivy [102] reported a case of successful osteotomy performed bilaterally on a markedly retruded, underdeveloped and ankylosed mandible.

For cleft palate, Brown [103] showed the feasibility of doing the push back operation with preservation of the arteries in one stage. A further development was the double push back in cases in which only scant tissue is available for lengthening the palate.

About three years ago, Ivy [104] undertook the repair of cleft palates by the Axhausen method. For cleft lip he preferred to operate on infants from 6 weeks to 3 months old, using the Veau anterior turnover flap from the vomer to close the anterior part of the cleft in the palate and following with closure of the cleft in the lip by the Blair-Mirault procedure. At 2½ to 3 years, the Axhausen operation is performed to close the remaining part of the cleft in the hard and soft palate. Reporting 57 cases in which the Veau-Axhausen sequence was followed, he stated that on follow-up results in 41 were recorded as good and results in 16 as fair—in general, better than obtainable with the classic Langenbeck operation. For patients with velopharyngeal insufficiency the push back operation of Dorrance, usually performed in two stages, not earlier than 4 years, has been highly successful.

In closing cleft lips, Brenizer [105] has found that fascia placed between the mucosa and the muscularis layer relieves tension on the suture line and produces a minimum of scarring. One of the most difficult parts of an operation for cleft lip is to secure a symmetric nostril on the affected side. Secondary operations to improve the nostril may be

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99. Kazanjian, V. H.: Congenital Absence of Ramus of the Mandible, Am. J. Orthodontics **26**:175-187 (Feb.) 1940.

100. Bellinger, D. H.: Temporomandibular Ankylosis and Its Surgical Correction, J. Am. Dent. A. **27**:1563-1568 (Oct.) 1940.

101. New, G. B., and Erich, J. B.: Retruded Chins, J. A. M. A. **115**:186-190 (July 20) 1940.

102. Ivy, R. H.: Anquilosis osea bilateral de las articulaciones temporomaxilares con una marcada deformidad facial, An. de cir. **6**:383-388 (Dec.) 1940.

103. Brown, J. B.: Double Elongation of Partially Cleft Palates and Elongations with Complete Clefts, Surg., Gynec. & Obst. **70**:815-818 (April) 1940.

104. Ivy, R. H.: Experiences in Cleft Palate Surgery, Ann. Surg. **112**:775-781 (Oct.) 1940.

105. Brenizer, A. G.: Skin and Fascia Grafting, Am. J. Surg. **47**:265-278 (Feb.) 1940.

necessary. Zeno's [106] technic is to expose the lower lateral cartilages through a butterfly incision in the under surface of the tip of the nose and trim them to corresponding size and shape. They are then sutured in this position.

A paper on the larynx by Figi [107] and one on cysts and fistulas of the thyroglossal duct by de J. Pemberton and Stalker [108] were of interest. Chronic stenosis of the larynx may be due to a variety of factors. The most difficult cases from a therapeutic point of view are those in which the stenotic condition was caused by perichondritis following irradiation of the tissues. If the scarring is extensive, open operation offers the best result. Correction consists of a midline opening of the trachea, complete excision of the scar tissue and lining with a thin skin graft which has been cut by the dermatome. The graft is introduced on a core of air foam sponge and sutured in place by silver wires brought out through the skin. The sponge is left in place ten days and is removed by means of a dilator.

Reporting 293 operations on abnormal thyroglossal ducts, de J. Pemberton and Stalker [108] related remarkably successful use of a modified Sistrunk operation for removal. In general, when a sinus is present, it is injected with methylthionine chloride (methylene blue), and an elliptic transverse incision is made to include the sinus. The cyst is dissected free; the central portion of the hyoid bone is resected, and the tissue up to the foramen cecum is coned out. The follow-up examination included 263 cases; in only 4 were recurrences noted.

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106. Zeno, L.: Deformidad nasal en el labio leporino, An. de cir. **6**:388-394 (Dec.) 1940.

107. Figi, F. A.: Chronic Stenosis of the Larynx with Special Consideration of Skin Grafting, Ann. Otol., Rhin. & Laryng. **49**:394-409 (June) 1940.

108. Pemberton, J. de J., and Stalker, L. K.: Cysts, Sinuses and Fistulae of the Thyroglossal Duct, Ann. Surg. **111**:950-957 (June) 1940.

News and Comment

HOME STUDY COURSES FOR 1941-1942

The American Academy of Ophthalmology and Otolaryngology will again offer its home study courses in the fundamentals of the two specialties, beginning August 1.

These courses were intended primarily for residents in specialized institutions that give no formalized instruction in fundamentals. The committee reports, however, that many physicians preparing for examinations given by special boards and many who were practicing in small communities without facilities for postgraduate instruction were included in the first year's registration of 465. A registration of about 150 had been expected.

Each registrant received on the first of each month a list of reading in standard textbooks designated by chapters. At the end of the month twenty questions covering this material were sent to the registrant. These were to be answered and returned to the committee. For each month one person was in charge of correcting and grading the papers, aided by a group of assistants, all diplomates of the special boards. Corrected papers were returned to the registrant.

The following topics were covered last year:

	Ophthalmology	Otolaryngology
August	Anatomy, histology, embryology	Anatomy I
September	Optics and visual physiology	Anatomy II
October	Physiology	Histology-pathology
November	Pathology	Physiology
December	Refraction	Bacteriology
January	Refraction	Biochemistry
February	Perimetry	Otolaryngologic examination
March	Muscles	Hearing tests
April	Biomicroscopy and ophthalmoscopy	Vestibular tests

The cost of the course is $10. The work is started August 1 in order that residents entering their service July 1 may have a month for orientation. The committee emphasizes that the course is for training in fundamentals and in no way equips a physician for clinical practice. Registration should be in the hands of Dr. William P. Wherry, 1500 Medical Arts Building, Omaha, by July 22.

Abstracts from Current Literature

Ear

TREATMENT OF ACUTE AND CHRONIC SUPPURATIVE OTITIS MEDIA. HAROLD I. LILLIE, J. A. M. A. **115**:506 (Aug. 17) 1940.

The author discusses suppurative otitis media, emphasizing the value of hospitalization, confinement to be in an uright position, application of hot moist dressings, maintenance of adequate warmth and moisture in the room and perhaps roentgenologic treatment in the early stages in order to relieve pain.

He comments on the controversy that prevails as to the exact time that myringotomy is indicated, but concludes with the statement that the treatment in each case should be individualized and that in the case in which palliative measures have been unavailing, myringotomy will give relief. He feels that it is best not to resort to irrigation of the auditory canal if the discharge from the ear is free—it should be done when the discharge becomes thick and purulent. Careful observations of the temperature, pulse and respiration must be charted, but the best index of the patient's general condition is his appearance and demeanor. The latter points are stated thus in two separate paragraphs:

"There is no set period for final resolution—every patient constitutes a law unto himself. The appearance is the most reliable guide. The question of surgical intervention (mastoidectomy) must be pondered in every individual case.

"Alcoholic solutions are best for local treatment and instillation in the ear, and all aural therapy is best carried out by an otologist."

The author considers four groups of patients with different clinical types of chronic suppurative otitis media, as follows:

"GROUP 1: Patients comprising this group include those in whom anteroinferior perforations of the tympanic membrane overlie the orifice of the eustachian tube. Few subjective symptoms are complained of other than discharge from the ear. The discharge usually is increased in the presence of head colds. The discharge is mucopurulent in character. . . . The treatment is directed at the nasopharynx, eustachian tube and middle ear." The patient is cautioned against forceful blowing of the nose.

"GROUP 2: Included in this group are those patients who may have had considerable central loss of the tympanic membrane and show evidence of definite pathologic change in the membrane covering the promontory. Hyperplasia and granulation tissue are frequently encountered." The use of drying solutions with certain antiseptic agents, drying with a stream of air and zinc ionization are the methods of treatment that give satisfactory results.

GROUP 3: Defects in the attic of various sizes and situation are seen. Occasionally partial or total destruction of the tympanic membrane may be present. Perforation is usually of the marginal type and the presence of cholesteatoma is to be expected. Necrosis of bone is not unusual. Subjective symptoms are pain, headache, mild attacks of vertigo and slight deafness, in addition to a malodorous discharge. Direct instrumentation can be done. The cases of these patients may be termed "borderline surgical," and the operation of choice, when and if it becomes necessary, is the modified radical mastoidectomy.

GROUP 4: These patients "present definite evidence of serious pathologic changes with or without symptoms or impending serious complication." Such cases call for surgical intervention to institute adequate drainage as well as to ward off any deeper ingress of infection that may threaten life. GORDON, Philadelphia.

PATHOLOGIC CHANGES IN THE PERIPHERAL AUDITORY MECHANISM DUE TO AVITAMINOSES (A, B COMPLEX, C, D AND E). WALTER P. COVELL, Laryngoscope **50**:632 (July) 1940.

Covell reports the histopathologic changes in the peripheral auditory mechanism of rats, guinea pigs and chicks fed diets deficient in one or another vitamin. The effects of deficiencies of vitamins A, B complex, C, D and E were studied. Middle ear infections were more frequent in the animals on deficient diets. With vitamin A deficiency there were keratinization and metaplasia of the epithelium of the middle ear, with secondary infection. Guinea pigs which were fed a diet deficient in vitamin C showed hemorrhage into the submucosa, with fibroblastic proliferation and sloughing. The muscles of the middle ears showed varying degrees of degenerative change in the fibers.

The membranous cochlea did not show marked degenerative changes for any of the avitaminoses. The cells of the spiral ganglions were mildly influenced in most of the deficiencies, more markedly in the B_2 deficiency. Demyelination of the distal portion of the cochlear nerve was in evidence in all the animals which had deficient diets, being greatest in those which lacked one of the factors of the vitamin B group.

Rats which were fed a diet deficient in vitamin A showed middle ear infections, periosteal thickening of the otic capsule, occasional exostoses in the internal auditory canal, degeneration of the cells of the stria vascularis and parts of the organ of Corti and some demyelination of the cochlear nerve. Animals which were fed a diet deficient in vitamin B showed degenerative changes in the external hair cells and demyelination of the dendritic portion of the cochlear nerve. Guinea pigs which were fed a diet deficient in vitamin C showed hemorrhage into the mucosa of the middle ear and swelling and degeneration of the organ of Corti. Rats which were fed a diet deficient in vitamin D showed osteoid laid down in the bones of the ossicular chain and the otic capsule and some demyelination of the cochlear nerve. Those which were fed a diet deficient in vitamin E showed fibrous replacement of the muscle bundles in the muscles of the middle ear, particularly the tensor tympani.

WOOD, Newark, N. J.

TRAUMATISM OF THE LABYRINTH BY ELECTRIC CURRENT. J. CALVET and R. LÔ, Oto-rhino-laryng. internat. **24**:5 (Jan.) 1940.

Calvet and Lô report 2 cases of injury to the labyrinth from a current induced in a telephone line by a nearby power line carrying 60,000 volts. The patients were 2 telephone workers, women of middle age. Both were rendered unconscious by the shock. The symptoms were typical of hysterotraumatic shock. Repeated examinations of the labyrinths revealed variations in the responses to tests. The patients remained incapacitated after eight years.

In a series of experiments on guinea pigs, to determine the pathologic changes produced by electric currents, Calvet and Lô observed numerous hemorrhages, involving especially the stria vascularis and the vestibular apparatus, and edema of Corti's organ. In addition to these lesions, with the attendant functional disturbances, there occurred later signs of deafness and vestibular disturbances, due to bulbar vasomotor lesions. The production of the peripheral lesions is attributed to an elevation of tension of the internal auditory artery, produced either by the electric shock, which is proportional to the intensity of the current, or by the accoustic shock, which is in relation to the intensity of the sonorous source.

DENNIS, San Diego, Calif.

SEVERE MASTOIDITIS. A. SARGNON, Oto-rhino-laryng. internat. **24**:225 (Aug.) 1940.

During the severe winter of 1939-1940 Sargnon observed 3 cases of severe mastoid infection. In 1 case the infection was complicated by superacute osteomyelitis, with spontaneous hemorrhage into the mastoid cavity. The second case

was that of a child with an infection due to Löffler's bacillus and phlebitis of the lateral sinus, and the third was that of a child in whom facial paralysis occurred forty-six hours after the onset of otitis. This patient was operated on immediately and made an unusually quick recovery. The other 2 patients also recovered.

DENNIS, San Diego, Calif.

APICITIS (PETROSITIS). PAUL FRENCKNER, Acta oto-laryng. **28**:1, 1940.

Frenckner reports 26 cases of petrous abscess, in 16 of which the abscess was drained through the concavity of the superior canal, which region in the solid angle he prefers to call the trigonum intercaniculare. Apicitis appears almost exclusively in well pneumatized pyramids. He found the pyramid pneumatized in more than one fourth of all the cases. The pyramid communicates with cell systems situated behind and to the side of the labyrinth by means of different cell passages, the superior, the posterosuperior, the inferior and the tractus subarcuatus, by means of which it can be infected and also drained. These different cell tracts pass by the labyrinth separately but often join to form a common cell system within the pyramid. In most cases one should be able to reach this apical infection through one of the upper cell passages, the tractus subarcuatus included, even if the infection did not enter just that way. According to certain authors, the upper cell passages and cell systems are the more common, and therefore it should be possible to reach those cell systems through the concavity of the superior canal, even though the infection did not pass into the apex in this region.

The subarcuate searching operation is meant to be applied partly in those cases in which a direct fistula is found within the concavity of the superior canal and partly also in those cases in which roentgen examination indicates the possibility that an inflammatory cell system is situated just behind the labyrinth and particularly behind the superior canal.

Frenckner's researches indicate beyond doubt that in those cases in which the pyramid is somewhat pneumatized a tractus subarcuatus has often developed or at least that a communication can exist at this point between the cell systems situated laterally and medially from the superior canal. Roentgen examination of 200 temporal bones in living persons reveals a probably accessible tractus subarcuatus in about a third of the cases. Macroscopic and microscopic examination of temporal bones from autopsy material indicates a still greater frequency.

Frenckner's method of approach is as follows:

1. A simple or radical mastoid operation is done, with careful removal of all accessible perilabyrinthine cells.

2. A careful search for a fistula is made; if one is found, it is followed.

3. If no fistula is found, he attempts to reach the concavity of the superior canal through the trigonum intercaniculare and then continues through a possible cell tract or goes carefully through the hard bone past the plane of the canal. If a free cavity is not found a few millimeters internal to the plane of the canal, this approach should be abandoned.

4. He then tries the approach of Ramadier.

Frenckner deprecates the use of an ordinary electric burr because of the danger of injuring the labyrinth by the friction-caused heat. This heat can be obviated by refrigerating the burr with solid carbon dioxide, but he prefers to use the curet.

GROVE, Milwaukee.

RESULTS OF SURGICAL REMOVAL OF ACOUSTIC TUMORS. VERZAR, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:129, 1939.

Verzar reports the results of 3 operations by the translabyrinthine method for the removal of acoustic tumors. The first patient died at the time of operation of respiratory failure. The second patient lived four months, and the third lived eight months.

The author prefers this method of approach to the tumors, since he was able in each case to enucleate the growth without any difficulty. He also avoids a large bony defect in the occipital region, with an opportunity for brain prolapse. He does not believe that opening of the mastoid cells has any definite significance in the course of the operation.

PERSKY, Philadelphia.

VESTIBULAR FINDINGS IN PATIENTS WITH ACOUSTIC TUMORS. UNTERBERGER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:133, 1939.

Unterberger describes the usual vestibular and cochlear findings in patients with acoustic tumors and stresses: (1) the variability in the direction and intensity of the nystagmus and its importance; (2) the postural nystagmus, a frequent finding in patients with these tumors, and its value, and (3) in the presence of induced stimulation (either rotational or caloric) the abnormal nystagmus, past pointing, falling, abnormal gait, arm weakness and head posture as evidences of abnormal responses of the head, back and extremities. However, he cautions against assuming that any of these findings are invariable, since he had 4 patients who presented typical clinical and vestibular symptoms of an acoustic tumor, yet at the time of operation the growth was found in the cerebellum or in the pons. The symptoms apparently were due to pressure, encroachment and, finally, increased intracranial pressure.

The diagnostic findings of an acoustic tumor are: (1) a characteristic nystagmogram; (2) definite abnormalities in induced labyrinthine reactions, which always conform to a certain type; (3) inability to hold the head up, arm droop and abnormal gait. The author stresses the difficulty in differentiating the findings associated with lesions of the brain stem and with destruction of the acoustic nerve.

PERSKY, Philadelphia.

HISTOLOGIC OBSERVATIONS IN A RECENT CASE OF RECESSIVE DEAF-MUTISM: DIFFERENTIATION OF THIS FORM OF DEAFNESS FROM THE ACQUIRED TYPE. W. ALBRECHT, Ztschr. f. Hals-, Nasen- u. Ohrenh. **46**:167, 1939.

Albrecht presented histologic specimens from a case of hereditary deaf-mutism of the recessive type. He found that the number and type of cells of the cochlear nucleus were normal. The structure, the volume and the course of the auditory nerve were within normal limits. No change was found in the bony structure of the cochlea. The entire organ of Corti was generally well formed except that some of the cells were missing. However, the stria vascularis showed some variations from the normal. In some of the turns of the cochlea, it was absent entirely, while in a few of the turns it was barely recognizable. A few strands of connective tissue were seen to stretch across the lumen from the wall of the ductus cochlearis. The upper turns of the ductus cochlearis appeared to be filled with a colloidal protein substance. The vestibular structures were not altered.

After a comparison of these findings with those seen in acquired deafness, the author believes that the changes in the organ of Corti are similar and that it is impossible to make a sharp differentiation between hereditary and acquired deafness.

PERSKY, Philadelphia.

Pharynx

HISTOLOGIC OBSERVATIONS ON THE BLOOD VESSELS AND THE LYMPHATIC NODULES AND ON THE EVENTUAL INCLUSION OF CARTILAGE AND BONE IN THE FAUCIAL TONSIL. G. ZANZUCCHI, Arch. ital. di otol. **52**:443 (Sept.) 1940.

The material for this study consisted of the tonsils removed at autopsy from 30 subjects who had presented no evidence of pathologic alterations of the tonsils. The subjects varied in age from 7 months of fetal life to 79 years. Attention was directed to the structure of the blood vessels, the fatty infiltration of the reticular cells of the lymph nodules and the inclusion of cartilaginous and osseous tissue in the tonsil.

For the study of the structure of the blood vessels, the material was divided into two groups: (*a*) arteries in the presumably normal tonsils and (*b*) arteries in the tonsils in involution. It is characteristic that in tonsils in which the lymphatic tissue does not present signs of regression the small arteries preserve their juvenile character to advanced age. In tonsils of this kind, elastic fibers in the vessel walls were scarce or entirely lacking. On the other hand, in those in which the lymphatic tissue was in regression and gave place to fibrous tissue, elastic fibers, even in the small arteries, were abundant in the intima, in the media and between the latter and the adventitia and soon showed a tendency to liquefaction. Thickening of the walls of the vessels and partial obliteration of the lumens proceed pari passu. Soon areas of fatty infiltration appear here and there. Therefore, it may be concluded that senile characteristics in the parenchymal arteries are manifested only in the areas of regression of the lymphatic tissue, never where the latter is conserved.

A characteristic finding, observed in all the subjects, independent of age, was the considerable lipoid content of the reticular cells in the clear centers of the lymph nodules. The probable explanation of this phenomenon is the assumption by these cells of fatty material diffused in the interstitial lymph, derived from the mass of fatty material contained in the tonsillar crypts. The fatty granules observed in the large globular cells of the clear centers probably represent fat derived from the degeneration of lymphocytes and phagocytosed by these cells.

The inclusion of cartilaginous and osseous tissue in the capsule and septums was observed in tonsils from subjects from 8 to 70 years of age. While primary ossification may occur, by far the most frequent process is chondrification, which may be followed by ossification. Zanzucchi discusses at length two theories of the source of the bone and cartilage: whether it is (*a*) an embryonic rest in the second branchial arch or (*b*) aberrant germs in the branchial skeleton. His conclusion, supported by a study of serial sections of the tonsils of 24 human fetuses, is that in all cases chondrification or the presence of osseous spicules depends on strictly local circulatory conditions. The claim that the cartilaginous nodules are supplied with perichondrium in children and not in the aged is not borne out by his observations. Osseous inclusions are sometimes independent from cartilaginous inclusions and are undoubtedly the result of membranous ossification. Many other times ossification occurs through metaplasia of the cartilaginous tissue, or more frequently, by substitution of the cartilage by bony tissue. The substitution proceeds directly inward from the perichondrium and not from the center toward the periphery.

DENNIS, San Diego, Calif.

Larynx

PATHOLOGY OF THE SUBEPITHELIAL (REINKE'S) LAYER OF THE VOCAL CORDS. RICHARD WALDAPFEL, Ann. Otol., Rhin. & Laryng. **49**:647 (Sept.) 1940.

In 1895 Reinke first described the area of the vocal cord which bears his name. This space is on the edge of the cord. The extent of the area above and below seems to be sharply limited at a line approximately where the glands appear. Edema of this area may occur in various processes in and surrounding the vocal cord. Polyps of the vocal cord seem to arise from the area. Tuberculosis of the cord can originate here and may remain temporarily limited in the space before spreading. Leukemic infiltration of the cord affects Reinke's space but only in its upper part adjacent to the ventricle. The same localization may be assumed for acute inflammation of the lymphoid tissue of the vocal cord. Carcinoma of the cords in its early stages develops only toward Reinke's space, and because of barrier-like boundaries and lack of lymph vessels in the area the space is not involved early. Thus the slow growth and the very late involvement of the lymph glands in carcinoma of the vocal cord are explained.

M. V. MILLER, Philadelphia.

CANCER OF THE LARYNX. MAX CUTLER, J. A. M. A. **115**:1339 (Oct. 19) 1940.

Cutler discusses the relation between gross anatomy, microscopic structure and radiosensitivity, basing his communication on a study of approximately 300 cases of cancer of the larynx encountered during the last ten years. His paper is illustrated with photographs and photomicrographs of lesions and with photographs of several patients after some therapy. Six case reports are given.

In his comments Cutler sets forth some principles. He states that the microscopic structure of a biopsy specimen is of some help in estimating prognosis and treatment but that its value is distinctly limited unless the microscopic changes are interpreted in the light of clinical and gross anatomic factors; else it may be misleading. Specimens for biopsy should be taken from different portions of the lesion. The radiosensitivity of a highly undifferentiated carcinoma may be predicted with reasonable certainty, but its radiocurability is dependent on several factors, e. g., the extent of the disease and the general condition of the patient.

In general, the prognosis for undifferentiated carcinoma (grades 3 and 4 of Broder's classification) is comparatively favorable under irradiation in spite of the great extent of the neoplasm, while that for differentiated carcinoma (grades 1 and 2) is most unfavorable in spite of the fact that the lesion is very small and limited in extent. The former are unsuitable for surgical treatment, while the latter are often curable by this means.

Cutler summarizes his paper as follows: "The main result of this investigation is the demonstration that the radiosensitivity of cancer of the larynx cannot be determined on the basis of microscopic structure alone. The most important indication of radiosensitivity is mobility of the lesion and surrounding structures."

Dr. Chevalier L. Jackson, in discussion, stated that the location of the lesion should guide the selection of treatment, that in his practice biopsy is done in every case and that in borderline cases the histologic features are taken into consideration. The best prognosis in cancer of the larynx, regardless of the treatment, probably lies with the better differentiated tumors.

GORDON, Philadelphia.

Nose

THE TREATMENT OF ACUTE AND CHRONIC SINUSITIS. J. MACKENZIE BROWN, J. A. M. A. **115**:508 (Aug. 17) 1940.

Brown discusses in some detail the treatment of sinusitis in children and adults, stressing the salient features of general treatment in the acute phases. These center about efficient drainage, the technic of instilling shrinking solutions, measures calculated to bring comfort, and drug therapy. Brown includes some comments on immunotherapy and chemotherapy.

Patients with sinusitis in the subacute phases, especially when free from pain and afebrile, respond best to thorough nasal shrinkage followed by gentle irrigation of the nose with warm Ringer's solution or to displacement treatment (Proetz).

Irrigation of the antrum is contraindicated in the acute stage, as a rule, and beneficial in the subacute. When acute maxillary sinusitis is due to dental pathologic conditions, without extraction, the antrum should be irrigated. When the acute process has subsided, the offending dental condition should be eradicated. Antral infection following dental extraction is best treated by irrigation through the socket if the latter connects with the antrum, but if no connection exists, the irrigation should be carried out through the nose.

The ethmoid and sphenoid sinuses, as well as the frontal sinuses, should respond to intensive shrinkage, which technic must be carefully and persistently carried out. If untoward symptoms arise, especially those indicating extension to the eye and/or intracranial structures, it is imperative to institute free drainage immediately as well as to remove as thoroughly as possible all infected cells. The only logical approach is by the external route.

In the treatment of chronic sinusitis, careful examination of the systems and regions in general and of the sinuses in particular is called for, including roentgen examination, allergic tests and metabolic studies. Local structural faults in the nose should be corrected, infected lymphoid elements in the pharynx removed and the nutrition of the patient balanced.

Chronic maxillary sinusitis should be treated by irrigation or if necessary by the making of an antral window. The presence of hyperplastic mucosa should suggest a check-up for the detection of allergy. In cases in which there are suppuration, hyperplasia and polyposis, a Caldwell-Luc operation is indicated. The same procedures should be carried out in those cases in which sinusitis is due to dental pathologic conditions.

Chronic disease of the ethmoid labyrinth may clear up when the antral disease is eliminated or after displacement treatment. If these measures are unsuccessful, an intranasal ethmoidectomy, with preservation of the middle turbinate, should be the method of choice. Failure to achieve good results might indicate the external approach.

Chronic disease of the sphenoid sinus will be helped in many instances by displacement treatment or by irrigation of the sinus through the natural opening, and if these measures prove unavailing, the making of an intranasal opening into the sinus should be undertaken.

Chronic frontal sinusitis calls for intranasal ethmoidectomy (Mosher). The remaining portion of the nasofrontal duct should not be interfered with. When orbital infection, fistula, osteomyelitic changes and other symptoms suggesting intracranial involvement become manifest, an external operation is indicated. In the operation no vestige of mucosa should be left and no bone should be left uncovered by periosteum.

The author concludes his paper with this summary:

"1. A careful general investigation of the patient's physical condition is advised in all types of sinusitis. This may disclose important underlying etiologic factors that may avert unnecessary local treatment or surgery.

"2. No surgical treatment should be undertaken during the acute stage unless urgency demands intervention.

"3. Conservation of the nasal mucosa is essential in all sinus surgery.

"4. The early recognition of serious complications with the proper treatment is imperative.

"5. Adequate drainage in both acute and chronic sinusitis is the greatest essential of treatment."

GORDON, Philadelphia.

OSTEOMYELITIS OF THE SKULL FOLLOWING FRONTAL SINUSITIS. A. S. MACMILLAN, J. A. M. A. **115**:1176 (Oct. 5) 1940.

Macmillan discusses in moderate detail the roentgenologic phases of osteomyelitis of the skull resulting from frontal sinusitis.

He stresses the necessity of examining the lateral view for the depth of the sinus and for the thickness of the anterior and posterior walls, as well as the importance of observing the relative thickness of the cortex to the medulla. Four views are taken as the minimum, i. e., a lateral and an anteroposterior view, the view with the patient in the Waters position while prone and the fluid level view.

It is well established that acute inflammation of the mucosa causes decalcification of the underlying bone, and in frontal sinusitis one must look for this decalcification.

Careful observation of the various views of the sinus must be made to determine the loss in density of bone, the loss of outline and the reaction in surrounding bone. It is often necessary to study composite pictures and to look for minute perforations of the anterior wall (due to small thrombosed vessels coming through the bone from infected mucous membrane).

In the 86 cases encountered by the author at the Massachusetts Eye and Ear Infirmary, the infection was limited to the front or posterior wall of the sinus.

It is important to realize that the bone is infected from 1¾ to 2 inches (4.5 to 5 cm.) beyond the necrotic area discernible on the roentgen film.

In fulminating osteomyelitis the one all-important sign was pitting edema of the forehead extending to the hair line.

This was present before any indication of involvement could be seen by roentgen examination. Microscopic examination of the bone removed at operation revealed thrombosis of infected veins.

Macmillan concludes as follows: "1. Osteomyelitis of the skull, because of its complications, is a most serious disease. 2. The x-ray evidence of this disease should not be waited for in the presence of clinical evidence. 3. Pitting edema to the hair line is a positive sign of infected bone. 4. Puttering surgery invites disaster."

GORDON, Philadelphia.

ALLERGIC TREATMENT OF CHRONIC SINUS CONDITIONS. MAURICE VAISBERG, New York State J. Med. **40**:631 (April 15) 1940.

Vaisberg thinks that most sinusitis is of allergic origin. He studied 50 cases over a three year period and produced marked relief in 10 per cent and complete relief in 72 per cent and the remaining patients were "helped." The investigation was extremely thorough, beginning with a complete medical history and running through the entire gamut of examination of eyes, ears, nose and throat, neurologic and laboratory tests, studies of skin sensitivity, serologic studies and other procedures. Reactions to intracutaneous injections of 0.01 cc. or 0.02 cc. in the forearms were "read" after twenty minutes, after forty-five minutes and after twenty-four hours, and as many as ninety tests were made at one sitting. A rigid basic diet was prescribed, with prohibition of all sensitizing foods, and also of inhalants, cosmetics and alcohol. The patient reported in two weeks, again in two weeks, and then, if improved, in three months. This rigid regimen usually brought definite relief, but a slump occurred at times. The cause was easily corrected, and relief then continued. A return to prohibited foods was gradual—one at a time until "immunity" was well established. The inference was derived that any cutaneous reaction, no matter how slight, may be of great general importance.

Every patient with "nose trouble" should be put through complete and exhaustive examination. "In chronic nasal and sinus affections, think of allergy first and operation last." But this regimen is difficult to carry out, and slight stuffiness or postnasal drip may be relieved more readily by the usual measures. One should select only cases in which the condition is severe, then obtain complete allergic control. If this is insufficient, immunologic control through chemotherapy (sulfanilamide when Streptococcus is present) or an autogenous toxoid filtrate must be obtained. Any surgical treatment should be limited to "very minor" procedures. "Major nasal and sinus procedures should be tried only as a last resort." Vaisberg says that 99 per cent of allergic conditions will "probably" clear up under strict adherence to the regimen he offers.

VOORHEES, New York.

DACRYOCYSTORHINOSTOMY BY WAY OF THE APERTURA PIRIFORMIS. F. J. MAYER, Monatschr. f. Ohrenh. **73**:760 (Dec.) 1939.

A technic inaugurated by Marschik, to approach the lacrimal sac by way of the piriform aperture, is described. After a mucous membrane flap similar to Halle's flap has been formed, a wedge with its point toward the aperture and its base toward the sac, is taken out of the bone of the aperture. In this way but little of the bony support which maintains the shape of the nose is lost. The tear sac is reached at its medial and mesioanterior surface. The infraorbital ridge remains intact, and the nasomaxillary suture is never crossed. The exposed tear sac is opened according to the previously known methods. The flap has to be remodeled so that it is not so large as to close the newly created opening for drainage of the sac. Iodoform gauze is used for packing. The author reports 60 cases in which he employed this technic with excellent results.

LEDERER, Chicago.

Miscellaneous

INDUSTRIAL ASPECTS OF EAR, NOSE AND THROAT PRACTICE. HAROLD A. FLETCHER, J. A. M. A. **115**:496 (Aug. 17) 1940.

Fletcher discusses in fair detail the various phases of industrial otologic practice and presents many interesting conclusions. His experience of over twenty years in this type of work imparts a note of authority to the many valuable points of information and advice.

The examiner should study the problem of any given case carefully with a view to determining whether any pathologic or anomalous condition is present, whether disability will result therefrom and whether correction of any fault is possible. Prejudice is unfortunate. Personal feeling should never enter into any case. The examiner should realize the varied types of personality and character among the patients and should never jump to any conclusions until all of the facts have been accumulated and due judgment has been exercised.

The specialist should be called early. A thorough history and the results of a careful examination, with comprehensive notes, constitute the most important data in any given case. Especial emphasis is placed on the history and the results of the first physical examination. Several examples are cited.

Nothing should be taken for granted, and examinations, as well as roentgen and laboratory tests, should be repeated if necessary.

Injuries to the face, nose and sinuses are fairly well understood and adequately treated. It is important to evaluate with care whether a given injury over a sinus may have caused sinal infection. Such a question is often hard to decide, and many factors must be considered before final conclusions are drawn. Examples are given of such cases. Other cases are cited proving the difficulty of evaluation in drawing conclusions.

Loss of hearing is fully discussed, and a number of illustrations are given of cases in which it is difficult to conclude with certainty just how much a given injury was the cause of deafness. Questions of gumma and the importance of vestibular tests are commented on.

It is often difficult to detect the malingerer who claims marked bilateral deafness, though the catch or confusion tests and especially the Stenger test will materially aid in many cases.

The author concludes with the thought that many of these problems are not recognized by as many otologists as they should be, that many points are controversial and that otologists should give the subject the benefit of group study and discussion to clarify these controversial points as much as possible.

(Dr. W. E. Grove, of Milwaukee, in the discussion states that Dr. Fletcher is "quite correct in saying that the most difficult question to evaluate after a head injury is the alleged loss of hearing.")

GORDON, Philadelphia

COLD VACCINES: A FURTHER EVALUATION. H. S. DIEHL, A. B. BAKER and D. W. COWAN, J. A. M. A. **115**:593 (Aug. 24) 1940.

Diehl and his co-workers review their results of over one year ago, when they compared observations on groups of students receiving, subcutaneously and orally, vaccines consisitng of mechanically killed organisms with observations on a control group given capsules filled with sterile physiologic solution of sodium chloride and lactose. The group of students receiving vaccine subcutaneously had 25 per cent fewer colds than did the control group, but it was deemed that this was not of enough practical importance to justify the time and expense involved in carrying out the intensive vaccination. The group receiving the oral vaccine experienced just as many colds as did the control group.

The present report consists of an evaluation of heat-killed bacterial vaccine. This vaccine contains the following in each cubic centimeter: Staphylococcus aureus and Staphylococcus albus, of each 1 billion organisms; Streptococcus, Pneu-

mococcus, Micrococcus catarrhalis, Friedländer's bacillus and influenza bacillus, of each 400 million. It was given as follows: 1. The first dose was 0.1 cc., with an increase of 0.1 cc. weekly for those patients having no reaction; the sixth and seventh doses were increased 0.25 cc. at two week intervals, and thereafter doses of 1 cc. were continued every two weeks throughout the season. 2. For students who had moderately severe local reactions to the vaccine the dose was not increased beyond that which produced the reaction, and this dose was continued at two week intervals throughout the season. 3. For students who had severe local reactions or moderately severe general reactions to the vaccine, the dose was decreased to the largest amount which did not produce a reaction, and this was continued throughout the season.

The control group, the members of which believed that they were receiving vaccine, were given hypodermic injections of sterile physiologic solution of sodium chloride at the same intervals that the experimental groups received the vaccine.

The authors concluded: "A carefully controlled study of the traditional heat-killed bacterial vaccine for the common cold reveals no evidence that it is of value in a group of cold-susceptible students at the University of Minnesota."

GORDON, Philadelphia.

ACOUSTIC TUMOURS. H. OLIVECRONA, J. Neurol. & Psychiat. **3**:141 (April) 1940.

The immediate, and especially the late, results of operation for acoustic tumors are hardly known. In summarizing his operative results in 130 cases of acoustic tumor from 1930 to 1939, Olivecrona finds that the immediate mortality rate in his series was 21.5 per cent and the remote mortality rate 8.8 per cent. Both the immediate and the remote mortality rate were lowest when the tumor was completely extirpated; subtotal extirpations had a less favorable prognosis, and intracapsular enucleations were followed by the highest mortality. Intracapsular enucleation gave excellent results only in cases of cystic or stationary tumor; otherwise, the neoplasm tended to recur. The postoperative morbidity rate, as measured in terms of capacity for work, showed in general that immediate return to full earning capacity was rare. The majority of patients with radical and subtotal extirpations were left with some defect which limited their capacity for work, but these defects gradually disappeared if sufficient time was allowed for convalescence and reeducation. The morbidity rate was lowest in the group of patients with intracapsular enucleations, but this was only apparent, since in this group patients with stationary tumors alone survived, the others having died of recurrence. With the development of better technic, Olivecrona gradually lowered the mortality rate for complete extirpation from 26.3 per cent, in the period from 1931 to 1933, to 11.1 per cent, in the period from 1937 to 1939; the frequent postoperative complication facial palsy also was prevented in 65 per cent of the cases. The author concludes that, with the exception of cystic tumors, complete removal of an acoustic tumor should be attempted in all cases.

MALAMUD, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

DEBARKATION SICKNESS. C. LARROUDÉ, Oto-rhino-laryng. internat. **24**:209 (July) 1940.

So-called channel fever is characterized by agitation, nervousness, general lameness, headache, anorexia, insomnia and nausea and vomiting. These symptoms appear at the end of a sea voyage, when land is already near. A disturbance with the same symptoms is described by Larroudé; it differs from channel fever by occurring only when the victim sets foot on land after a sea voyage. An illustrative case is presented, that of a patient who in the course of his first long sea trip, during which he had no twinge of seasickness, became ill as soon as he set foot on land. This experience was repeated at three points where landings were made.

As soon as he reembarked, relief was experienced. On arrival at his destination he was incapacitated for four days. A probable hypothesis for the cause is that the patient's sensitive vestibular system became gradually adapted to the movements of the vessel during the voyage and on landing the sudden cessation of the vestibular stimuli produced a hypervagotonic crisis. DENNIS, San Diego, Calif.

HEALING IN PEMPHIGUS OF THE UPPER RESPIRATORY TRACT WITH GERMANIN. P REHÁK, Monatschr. f. Ohrenh. **12**:745 (Dec.) 1939.

After a discussion of present knowledge of pemphigus, Rehák reports 3 cases in which treatment was with germanin. According to the advice of Veiel, 0.5 Gm. is given at the first injection, then 1 Gm. every other day until four injections have been given. The patients reported as cured had lesions in the region of the nasopharynx, hypopharynx and epiglottis. LEDERER, Chicago.

RHYTHMIC AND SYNCHRONOUS MYOCLONIAS OF THE PHARYNX AND LARYNX. A. KREINDLER, Schweiz. Arch. f. Neurol. u. Psychiat. **43**:79, 1939.

Rhythmic myoclonis of the palate and pharynx in the great majority of previously reported cases have been due to vascular accidents. Most of the patients have been older persons. The condition has been attributed to a lesion of the central tegmental tract with pseudohypertrophic degeneration of the corresponding inferior olive. In cases in which the central tegmental tract was not involved, a lesion of one of the dentate nuclei has been associated with pseudohypertrophic degeneration of the contralateral inferior olive. The most striking clinical features are the constancy of rhythm and the fact that all the muscles participating in the myoclonic movements contract synchronously.

A woman aged 63 suffering with hypertension had quickly recovered from right hemiplegia three years previously. Symptoms of pseudobulbar palsy had developed after a second vascular accident, with left hemiplegia, six months before. Whenever the patient looked to the right, rhythmic movements of the eyeballs were observed. These movements were not on the order of nystagmus, as the slow and quick components characteristic of the latter condition were lacking. Synchronous with the movements of the eyeballs were other myorhythmias in the lower half of the face on the right side, the right sternocleidomastoid muscle, both sides of the soft palate and the posterior wall of the pharynx. Muscles of the floor of the mouth, i. e., the digastric and mylohyoid muscles, were likewise involved, and up and down movements of the thyroid cartilage were noted. Of the intrinsic musculature of the larynx, only the arytenoid muscles showed myoclonic movements.

Graphic records of the myoclonic movements of the right side of the face and the right sternocleidomastoid muscle were obtained with the use of Marey tambours. Movements of the palate were recorded by means of a small balloon placed within the patient's mouth and connected with a tambour. Muscles studied in this manner were found to contract with a synchronous rhythm varying from 85 to 110 contractions per minute. The individual movements consisted of a quick contraction and a slower relaxation, which was followed after a short pause by a slower contraction in the opposite direction. The movements did not cease during the act of swallowing; their amplitude was greater during inspiration than during expiration, and their frequency could be increased by pressure over the carotid sinus.

Persistence of the myoclonic movements during the act of swallowing is believed to indicate that rhythmic impulses responsible for the movements are conducted to the bulbar nuclei from higher centers but not by way of the pyramidal tracts. Significance is attached to the fact that the sucking movements of nurslings have a rhythm of 80 to 90 per minute, which approaches closely the rhythm of the myoclonic movements which have been described. Both types of movement, furthermore, involve similar groups of muscles. Kreindler and his associates have shown that the excitability of vestibular centers can be altered by pressure over the carotid sinus. It would appear, therefore, that stimulation of the carotid sinus has an influence on several, perhaps all, of the bulbar centers.

The medulla oblongata is the seat of certain automatisms essential for life, i. e., circulation of the blood and respiration. It seems likely that centers for the control of the musculature of the pharynx, larynx, face and eyes have a similar capacity for automatic and rhythmic activity but under normal conditions are inhibited in this respect. The view is expressed that a lesion either of a supranuclear center or of a tract leading to the centers for control of these muscles releases this automatic activity and thus produces the myoclonic movements.

Automatic activity, which has the characteristics of rhythmicity and periodicity, is a property of centers scattered throughout the central nervous sysem. It is not necessarily induced by afferent impulses. Most ganglion cells, in fact, have a rhythmic activity peculiar to themselves, the synchronized activity of great cell masses being well illustrated by electroencephalographic tracings. Kreindler and his co-workers have observed rhythmic movements of the tail in decerebrated cats. Action currents obtained from the isolated medulla oblongata of fishes show a rhythm similar to that of respiration. The respiratory center functions automatically under normal conditions, while other centers betray their capacity for automatic activity only when released from the control of higher levels.

DANIELS, Denver [ARCH. NEURO. & PSYCHIAT.]

PROGNOSIS AND TREATMENT OF RHINOGENOUS AND OTOGENOUS BRAIN ABSCESSES. ARNE A. SJÖBERG, Acta oto-laryng. **27**:638, 1939.

On the basis of two recent brain abscesses handled at the otolaryngologic clinic of Sabbatsberg Hospital, in Stockholm, Sweden, Sjöberg reviews the statistics of otitic and rhinologic brain abscesses encountered at this clinic from 1912 to the present time.

CASE 1.—After having a headache for some time, a 33 year old man with chronic otitis and cholesteatoma suddenly became unconscious, with signs of perforation meningitis. At operation an extradural abscess was found, which had induced a cerebellar abscess. After wide incision of the cerebellar abscess, with diathermic treatment and drainage and intensive use of azosulfamide (disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′, 6′-disulfonate) (administered orally, intramuscularly and intraspinally), the meningitis was checked and the patient recovered.

CASE 2.—A 19 year old youth became ill with an acute frontal sinusitis on the left side and a subperiosteal perforation to the upper eyelid. He was subjected to an external operation on the frontal sinus and was discharged free of symptoms. Six weeks after the onset of the illness, acute symptoms of an abscess of the left frontal lobe appeared. This abscess was drained by means of a diathermic incision of its wall, and a rubber tube was sewed to the skin. After two months the patient was discharged free of symptoms.

At the Sabbatsberg clinic there were in the years 1912 to 1938 24 cases of abscess of the temporal lobe, with a mortality of 87.5 per cent. During the same period there were 10 cases of cerebellar abscess, with a mortality of 50 per cent. The abscesses of both groups were complications of acute otitis.

In 18 of the cases of chronic otitis, abscess of the temporal lobe occurred, with a mortality of 61.1 per cent, and in 13 cases there was cerebellar abscess, with a mortality of 46.1 per cent.

The mortality in all the cases in which brain abscess followed acute otitis was 76.5 per cent and in all the cases in which brain abscess followed chronic otitis, 51.6 per cent.

During the same period there were 8 cases in which abscess of the frontal lobe followed acute sinusitis and 8 in which such an abscess followed chronic sinusitis. Only 1 of the 8 patients with acute sinusitis recovered, while 3 of the 8 with chronic sinusitis recovered, giving a combined mortality from all abscesses of the frontal lobe of 75 per cent. It therefore appears that the mortality in all

cases of rhinologic brain abscess is higher than that in all cases of otologic brain abscesses and that the prognosis in cases in which there is frontal lobe abscess of acute origin is somber.

In the Sabbatsberg clinic there was about 1 brain abscess among 193 cases of otitis. The statistics of that institution show that cerebral abscesses outnumber the cerebellar in the proportion of 2:1 (42:23).

The statistics of most other clinics indicate that brain abscess is more frequent after chronic than after acute otitis. In the Sabbatsberg clinic brain abscess is somewhat more frequent after acute otitis than after chronic otitis.

Heine and Beck, on the basis of a large collection of cases from the literature, give a mortality for brain abscess of 74.17 per cent. Sercer gives 78 per cent; Meuerman, 69.3 per cent, and the Sabbatsberg clinic, 66.1 per cent.

In the presence of rhinologic or otologic brain abscess, surgical therapy should not be begun until the abscess is encapsulated, at the earliest, two to three weeks after the onset. The pressure on the brain and not the infection is the deciding factor for the choice of the time. The amount of pressure on the brain is determined by observation on the degree of bradycardia, daily examination of the fundi oculorum and repeated tests of the field of vision. Encephalographic examination may be necessary to locate the abscess, but the making of lumbar punctures before the evacuation of pus may involve risks.

When the abscess is to be evacuated, a longitudinal incision is first made in the dura by diathermy, then the puncture is made and the pus removed slowly by cannular suction. Next, a wide incision of the wall of the abscess is made by diathermy and the cavity is inspected by means of a nasal speculum. A coarse rubber drain is inserted into the cavity, sewed to the skin and left in place five to six weeks. If meningitis threatens, sulfanilamide is administered by mouth, intramuscularly and intraspinally.

GROVE, Milwaukee.

Book Reviews

The Ear, Nose and Throat. By Dr. Franz Conde Jahn, professor of clinical otorhinolaryngology, National Academy of Medicine. Paper. Printed in folio, 248 pages. Caracas, Venezuela, 1941.

This volume presents a rational discussion of the common ailments of the ear, nose and throat. Symptomatology, prognosis and medical treatment are stressed. The indications for surgical intervention are also clearly outlined. A considerable amount of emphasis is placed on dietary and hygienic regimen, physical therapy hydrotherapy and climatotherapy.

While most of the common ailments are discussed, it seems that emphasis is placed on the routine follow-up treatment as applicable particularly to general practice. Little emphasis has been placed on anatomy, physiology and surgical procedure. However, the discussions of each malady from the prognostic and medical therapeutic standpoints are rational and in accord with prevailing opinions.

This is a splendid handbook for the general practitioner and should be of much interest even to the otorhinologist, since so much stress has been laid on the hygienic and medical problems.

The book is published in Spanish.

Manual of Clinical Chemistry. By Miriam Reiner, M.Sc., assistant chemist in the Mount Sinai Hospital, New York. Introduction by Harry Sobotka, Ph.D., chemist in the Mount Sinai Hospital, New York. Price $3. Pp. 296. New York: Interscience Publishers, Inc., 1941.

This manual represents the efforts of the author to describe in the smallest possible space the important tests and determinations of the chemical constituents of the body fluids, secretions and excretions. She has succeeded well in her purpose.

One method for each contingency is given. The method selected for any given determination is the one which is used most frequently in the hospital laboratory or which has come by common usage to be preferred by clinical chemists. It also is the simplest as well as the most economical. The bibliography is clearly indicated at the head of each method outlined. The text is further enhanced by two indexes, one a subject index, the other an author index.

The chapters discuss blood analysis, chemotherapy, urine, cerebrospinal fluid, feces, toxicologic tests, gastric analysis, function tests, sex hormones, vitamins and other subjects.

All procedures are clearly described with such simplicity that, after a little training, any one should be able to make accurate determinations.

This volume will be found a handy reference book by the laboratory worker and technician as well as the clinical pathologist. The otolaryngologist should find some points of interest because the manual bridges certain gaps between biochemistry and clinical medicine.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
President-Elect: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-24, 1941.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.

American Laryngological Association

President: Dr. T. E. Carmody, 227–16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Ave., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Henry P. Johnson, 32 Deering St., Portland, Maine.

Southern.—Chairman: Dr. William L. McDougall, 915 Doctors' Bldg., Atlanta, Ga.

Middle.—Chairman: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis, Mo.

Western.—Chairman: Dr. Emil F. Tholen, 1136 W. 6th St., Los Angeles.

American Otological Society

President: Dr. E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.